Rethinking the Dental Model

Reversing current downward trends in income may entail a change in the way we practice dentistry.

In May, I attended a conference in Albany held in conjunction with a NYSDA-sponsored meeting of the president elects of the Association’s components. The title of the conference was “The Changing Dental Care Landscape.” The moderator was Marko Vujicic, Ph.D., managing vice president of the ADA Health Policy Institute. The ADA recently completed a study, headed by Dr. Vujicic, concerning the future of dental practice in the United States. What the study uncovered is eye-opening.

The current downturn in dental spending didn’t just happen. It began around 2002. At the beginning of the recession, dental spending flattened out; it has yet to recover to pre-recession levels. As a consequence, incomes of dentists have declined. A disturbing aspect of this is that these income figures show no sign of improving, as has happened in the past.

Driving this downturn has been a decrease in dental usage by people 19 to 64 years of age. Conversely, children’s dental use has actually increased, as has that of the elderly. The increase in pediatric dental services is due to a rise in Medicaid eligibility. On the opposite side of the spectrum, children from more affluent families haven’t curtailed dental visits, but they also haven’t increased them significantly.

Why the downturn in the middle-age group? It basically comes down to needs vs. wants. Since dentistry is more discretionary-income driven, it appears that this age group prefers to spend money on other things. Its members also tend to be healthier. And they don’t see the need for regular dental care if they are not experiencing symptoms of a problem. Moreover, people in the middle tend to have more constraints on their time. They don’t, or perceive they don’t, have time to go to the dentist.

Add to this the fact that there has been a decrease in the amount of dental insurance coverage available to this group, either privately or as part of their compensation packages at work. Poorer individuals do not have the income needed to pay for dental services, and only a few states offer adult Medicaid coverage.

People in the middle have also seen sluggish growth in their personal incomes, leading to more and more adults saying they need dental services but can’t afford them. This in turn has led to an increase in emergency room dental visits. Over the past 10 years, the number of visits to an emergency room for dental problems has doubled, an increase driven by people 21 to 35 years old. Unfortunately, their ER visits rarely attack the problem that caused the visit in the first place. Treatment is usually palliative, aimed at getting the patient out of pain, not at correcting the problem.
All of this change in spending has hurt dentists financially. Practice revenues are down on average for both generalists and specialists. Looking ahead, we see a shift in emphasis on value. What value will we give our patients with the treatments we render? Will patients perceive this value? What are the appropriate quality values in dentistry? It seems it will take some time to determine exactly what this means and how we can control the outcome. But control it we must, or we will be bulldozed into irrelevance.

There is a movement in medicine away from the solo practitioner, or silo, mentality toward the team approach. Dentistry is still a cottage industry. Most of its practitioners work alone or in partnership arrangements. Even though the corporate model is beginning to grow, most dental offices don’t operate that way. Meanwhile, physicians are moving into larger groups with more specialists as part of the group. Dr. Vujicic recommended that dentistry follow this trend. He sees larger group practices in the future, with all specialists represented in them. And, he predicts, as with medicine, participants will be rewarded by positive outcomes and not just by rendering treatment.

### OFFICERS

<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>John J. Liang, President</td>
<td>2013 Geneva St., Utica, NY 13501</td>
</tr>
<tr>
<td>David J. Miller, President Elect</td>
<td>467 Newbridge Rd., East Meadow, NY 11554</td>
</tr>
<tr>
<td>Richard F. Andolina, Vice President</td>
<td>74 Main St., Hornell, NY 14843</td>
</tr>
<tr>
<td>Mark J. Weinberger, Treasurer</td>
<td>78 Southbury Rd., Clifton Park, NY 12065</td>
</tr>
<tr>
<td>Robert M. Peskin, Speaker of the House</td>
<td>601 Franklin Ave., #225, Garden City, NY 11530</td>
</tr>
<tr>
<td>Mark J. Feldman, Executive Director</td>
<td>20 Corporate Woods Blvd., Albany, NY 12211</td>
</tr>
<tr>
<td>Steven Gounardes, ABA Trustee</td>
<td>351 87th St., Brooklyn, NY 11209</td>
</tr>
</tbody>
</table>

### BOARD OF TRUSTEES

<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joel M. Friedman, Immediate Past President</td>
<td>525 E. 60th St., New York, NY 10065</td>
</tr>
<tr>
<td>NY – Edward J. Miller Jr</td>
<td>121 E. 60th St., #7A, New York, NY 10022</td>
</tr>
<tr>
<td>2 – James Sconzo</td>
<td>1646 Marine Pkwy, Brooklyn, NY 11234</td>
</tr>
<tr>
<td>3 – Lawrence J. Busino</td>
<td>2 Executive Park Dr., Albany, NY 12203</td>
</tr>
<tr>
<td>4 – Frederick W. Wetzel</td>
<td>1556 Union St., Schenectady, NY 12309</td>
</tr>
<tr>
<td>5 – William H. Karp</td>
<td>4846 4500 Pavon Lane, Morris, NY 13104</td>
</tr>
<tr>
<td>6 – Scott J. Farrell</td>
<td>39 Loney St., Binghamton, NY 13905</td>
</tr>
<tr>
<td>7 – Robert J. Buhite II</td>
<td>544 E. Ridge Rd., #201, Rochester, NY 14621</td>
</tr>
<tr>
<td>8 – Brendan Dowd</td>
<td>6932 Williams Rd., #1900, Niagara Falls, NY 14004</td>
</tr>
<tr>
<td>9 – Anthony Cuomo</td>
<td>667 Shoreleigh Ave., #201, Carmel, NY 10512</td>
</tr>
<tr>
<td>N – Michael S. Shreck</td>
<td>1300 Union Turnpike, #201, New Hyde Park, NY 11040</td>
</tr>
<tr>
<td>Q – Joseph R. Caruso</td>
<td>40-29 Utopia Pk., Aquebogue, NY 11508</td>
</tr>
<tr>
<td>S – Robert P. Leary</td>
<td>80 Maple Ave., #206, Smithtown, NY 11787</td>
</tr>
<tr>
<td>B – Richard P. Herman</td>
<td>2 Lockwood Lane, New Windsor, NY 12553</td>
</tr>
</tbody>
</table>

### COUNCIL CHAIRPERSONS

<table>
<thead>
<tr>
<th>Council on Awards</th>
<th>Steven Gounardes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dental Benefit Programs</td>
<td>Viren Jhaveri</td>
</tr>
<tr>
<td>Dental Education &amp; Licensure</td>
<td>Anthony Ficara</td>
</tr>
<tr>
<td>Dental Health Planning &amp; Hospital Dentistry</td>
<td>Carl H. Tegtmeier</td>
</tr>
<tr>
<td>Dental Practice</td>
<td>Paul Abicocco</td>
</tr>
<tr>
<td>Ethics</td>
<td>Richard B. Serchuk</td>
</tr>
<tr>
<td>Governmental Affairs</td>
<td>Laura Medrano</td>
</tr>
<tr>
<td>Membership &amp; Communications</td>
<td>Jay Skolnick</td>
</tr>
<tr>
<td>Peer Review &amp; Quality Assurance</td>
<td>Egidio Farone</td>
</tr>
<tr>
<td>Professional Liability Insurance</td>
<td>David Delaney</td>
</tr>
</tbody>
</table>

### OFFICE

<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mark J. Feldman</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Lance Plunkett</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Beth M. Waneck</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Michael J. Herrmann</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Judith L. Shub</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Joshua Poupore</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Laura B. Leon</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
<tr>
<td>Mary Grates Stoll</td>
<td>476 AlbanyShaker Rd., Loudonville, NY 12211</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>Executive Director</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>General Counsel</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>Associate Executive Director</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>Assistant Executive Director Finance-Administration</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>Assistant Executive Director Health Affairs</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>Assistant Executive Director Marketing and Communication</td>
<td>Mark J. Feldman</td>
</tr>
<tr>
<td>Managing Editor</td>
<td>Mary Grates Stoll</td>
</tr>
<tr>
<td>Managing Editor</td>
<td>Mary Grates Stoll</td>
</tr>
</tbody>
</table>

The New York State Dental Journal  •  JUNE/JULY 2014 5
All is not lost, though. There will be plenty of opportunities out there for dentists who can adapt to future changes. Dr. Vujicic likened this to how Wayne Gretzky played hockey. Gretzky was such an accomplished player because he always anticipated where the puck would be, not where it was at the time. The same will be true in dentistry. We can’t continue to chase yesterday’s challenges. We must anticipate what the future challenges will be and address them now. Toward that end, the ADA has established its Center for Professional Success, a go-to resource to assist dentists in their practices and professional lives.

Dentists need to rethink how they dispense care. They must put the value of the service at the forefront and think about the best way to deliver that value. It may be something as simple as revisiting the “see your dentist twice a year” dogma we have all accepted during our professional lives. We must adjust the treatment to fit the patient’s needs. Some patients may need more frequent visits, while others need to be seen less often. Patients must be able to see the value in what we provide and not just the price tag associated with it.

Dentists must leverage the value agenda. They need to consolidate their services and measure their practices. Analytics is the big thing in sports today. Every team measures all sorts of data. Dentists need to do the same. After all, if you don’t know what you are doing, and how you are doing it, you can’t respond to change.

We need to interact more with our medical colleagues. Physicians know close to nothing about the oral cavity. We can help correct this situation. Screening for chronic diseases in the dental office through sialography is becoming an important part of dental practice. This can be a win/win for the dentist and the physician. The patient gets screened in the dental office and gets referred to the physician for any disease discovered.

Physicians need to understand the important relationship between the oral cavity and a patient’s overall health. We know this, but they don’t. We must educate them. Many diseases have oral components. Sometimes the first indication of a systemic problem appears in the mouth. With good communication with our physician colleagues, we build trust and, through that trust, are able to treat our patients more completely.

We must be more proactive in our approach to the future. The dental landscape is changing, and the challenges that lie ahead are formidable. This is a seminal moment for dentistry. How we react to the changes coming will determine if we are in control or if we will be controlled. I definitely want to be on the side of control. How about you?
CONTRIBUTORS

Root Amputation
A New Look into an Old Procedure
Rania Livada, D.D.S., M.S., is assistant professor in the Department of Periodontology, College of Dentistry, University of Tennessee Health Science Center, Memphis, TN.

Norman Fine, D.M.D., M.S., is in private practice limited to periodontics and implants in Greenville, SC.

Jacob Shiloah, D.M.D., is professor in the Department of Periodontology, College of Dentistry, University of Tennessee Health Science Center, Memphis, TN.

Management of the Dental Patient on Anticoagulant Medication
A Review
Manoj Agarwal, M.D.S., is assistant professor, Department of Conservative Dentistry and Endodontics, Government Dental College and Hospital, Jaipur, Rajasthan, India.

Sankalp Mittal, M.D.S., is assistant professor, Department of Oral and Maxillofacial Surgery, Government Dental College and Hospital, Jaipur, Rajasthan, India.

Sharmistha Vijay, M.D.S., is associate professor, Department of Periodontology and Implantology, Government Dental College and Hospital, Jaipur, Rajasthan, India.

Pooja Yadav, M.D.S., is assistant professor, Department of Prosthodontics, Mahatma Gandhi Dental College and Hospital, Jaipur, Rajasthan, India.

Vasim Raja Panwar, B.D.S., is a research fellow.

Neha Gupta, M.D.S., is assistant professor, Department of Prosthodontics, Government Dental College and Hospital, Jaipur, Rajasthan, India.

Angiofibrolipoma of the Retromolar Pad Region
Case Report with a Review of Literature
Farzaneh Agha-Hosseini, D.D.S., M.Sc., is professor in the Department of Oral Medicine, Dentistry School, Tehran University of Medical Sciences, Tehran, Iran.

Elham Moslemi, D.D.S., M.Sc., is professor in the Department of Oral Medicine, Dentistry School, Tehran University of Medical Sciences, Tehran, Iran.

The Residual Radicular Cyst
Jessica Lee, D.D.S., is an oral and maxillofacial surgery resident at NYU Langone Medical Center, Bellevue Hospital Center, New York, NY. She is a former research assistant in the Salivary Gland Center, Columbia University College of Dental Medicine, New York, NY.

John Costandi, D.M.D., is an oral and maxillofacial surgery resident at New York-Presbyterian Hospital (Columbia campus), New York, NY.

Louis Mandel, D.D.S., is director of the Salivary Gland Center and associate dean and clinical professor, oral and maxillofacial surgery, Columbia University College of Dental Medicine, New York, NY.

Treatment of a Mandibular Cyst Before Implant Placement
Case Report
Miles Yacker, D.D.S., is clinical associate professor and course director of implant dentistry: Biological Basis and Implant Surgery Seminar in the Ashman Department of Periodontology and Implant Dentistry, New York University College of Dentistry, New York, NY, and in private practice in Lawrence, NY.
John Ricci, Ph.D., is associate research scientist in the Department of Biomaterials and Biomimetics, New York University College of Dentistry, New York, NY.

Ioana Chesnou Matei, D.D.S., M.S., is a resident in postgraduate periodontics, Ashman Department of Periodontology and Implant Dentistry, New York University College of Dentistry, New York, NY.

Bin Hu, M.D., is associate research scientist in the Department of Biomaterials and Biomimetics, New York University College of Dentistry, New York, NY.

Sachin Mamidwar, M.B.B.S., M.S., is general manager of Orthogen, LLC, Springfield, NJ.

Oral and Dental Manifestations of Celiac Disease
William James Maloney, D.D.S., is a clinical associate professor in the Department of Cariology and Comprehensive Care at New York University College of Dentistry, New York, NY.

George Raymond, D.D.S., is a clinical instructor in the Department of Cariology and Comprehensive Care, New York University College of Dentistry, New York, NY.

David Hershkowitz, D.D.S., is clinical assistant professor and associate chair, Department of Cariology and Comprehensive Care, New York University College of Dentistry, New York, NY.

Glenn Rochlen, D.D.S., is clinical assistant professor, Department of Cariology and Comprehensive Care, and group practice director, New York University College of Dentistry, New York, NY.

The Plasma-Rich in Growth Factor as a Suitable Matrix in Regenerative Endodontics
A Case Series

Hengameh Bakhtiar, D.D.S., M.Sc., is assistant professor in the Department of Endodontics, Tehran Dental Branch, Islamic Azad University, Tehran, Iran.

Mehdi Vatanpour, D.D.S., M.Sc., is assistant professor in the Department of Endodontics, Tehran Dental Branch, Islamic Azad University, Tehran, Iran.

Arezoo Rayani, D.D.S., is a general dentist in Tehran, Iran.

Fina Navi, D.D.S., M.Sc., is assistant professor in the Department of Endodontics, Tehran Dental Branch, Islamic Azad University, Tehran, Iran.

Ehsan Asna-Ashari, D.D.S., M.Sc., is assistant professor in the Department of Endodontics, Tehran Dental Branch, Islamic Azad University, Tehran, Iran.

Anahid Ahmadi, D.D.S., is a general dentist in Tehran, Iran.

Hamid Jafarzadeh, D.D.S., M.Sc., is associate professor in the Dental Research Center, Department of Endodontics, Faculty of Dentistry, Mashhad University of Medical Sciences, Mashhad, Iran.
The New York State Department of Health (DOH) has prioritized tobacco cessation as a public health initiative. The department now includes coverage for smoking cessation counseling (SCC) for Medicaid patients by dental practitioners. This policy is consistent with public health recommendations from numerous sources, including the American Dental Association. The associated Medicaid policies are included in the May 2014 DOH Medicaid Update, available on the DOH website, www.health.ny.gov.

Dental practitioners will be able to provide and receive Medicaid reimbursement for SCC services as defined in their scope of practice. There have been changes to New York State law defining the scope of practice of dentistry since 1992, including inclusion of the term “maintaining dental health” in the scope of practice and continuing education requirements on the topic of the adverse health effects of tobacco use. Based on these changes, the State Board for Dentistry has determined that prescribing smoking cessation products is currently within the scope of practice of dentistry. The DOH’s expansion of Medicaid benefits does not supersede or alter any regulations issued by the New York State Department of Education governing scope of practice for dentists or dental hygienists.

Medicaid Coverage for SCC
Coverage for SCC in Medicaid became effective April 1 in the fee-for-service Medicaid program and on July 1 for the Medicaid managed care program. Dental practitioners will be allowed to provide two SCC sessions to a Medicaid beneficiary within any 12 continuous months. NYSDA advises dentists who provide treatment to Medicaid recipients enrolled in managed care to review their contracts to determine how this change affects their reimbursement and whether any changes need to be made to their participating provider contracts.

According to DOH, reimbursement to office-based dental practitioners will be at the rate of $10 per visit. To be eligible for reimbursement, claims must meet the following criteria:
- SCC must be provided face-to-face by either a dentist or a dental hygienist who is supervised by a dentist.
- SCC must be billed by either an office-based dental practitioner or by an Article 28 clinic that employs a dentist.
- Dental practitioners can only provide individual SCC services. There is no reimbursement for SCC provided to groups of patients in one session.
- SCC must be greater than three minutes in duration.
- Dental claims for SCC must include the CDT procedure code D1320 (tobacco counseling for the control and prevention of oral disease).
- Article 28 clinics that bill based on ambulatory patient group models must include the ICD-9 CM Diagnosis code 305.1 (tobacco use disorder).
- In a dental office or an Article 28 clinic, SCC should only take place during a dental visit as an adjunct when providing a dental service. It cannot be billed as a stand-alone service.
- A dental practitioner will be allowed to provide two smoking cessation counseling sessions to a Medicaid beneficiary within any 12 continuous months.
- SCC complements existing Medicaid-covered benefits for prescription and non-prescription smoking cessation products, including nasal sprays, inhalers, Zyban (bupropion), Chantix (varenicline), over-the-counter nicotine patches and gum. (Bear in mind that dental hygienists cannot prescribe medication to patients.)

To receive reimbursement for SCC services, the following information must be documented in the patient’s dental record:
1. At least four of the “5 A’s” as described below.
2. If patient is willing to quit, an offer of medication as needed, a target date for quitting and a follow-up date—with documentation in the record that the follow-up occurred.
3. If patient is unwilling to quit, the patient’s expressed roadblocks.
4. Referrals to the New York State Smoker’s Quitline and/or community services to address roadblocks and for additional cessation resources and counseling, if needed.

Smoking Cessation Guidelines
DOH’s criteria for Medicaid reimbursement for SCC are based on two guidelines: the “5 A’s of treating tobacco dependence” and the “5 R’s.” DOH cites the Clinical Practice Guideline “Treating Tobacco Use and Dependence: 2008 Update” that demonstrated that efficacious treatments for tobacco users exist and recommends that prac-
tioners follow the “5 A’s” of treating tobacco dependence. They are:
1. Ask the patient about tobacco use at every visit and document the response.
2. Advise the patient to quit in a clear and personalized manner.
3. Assess the patient’s willingness to make a quit attempt at this time.
4. Assist the patient to set a quit date and make a quit plan. Offer medication as needed.
5. Arrange to follow up with the patient within the first week, either in person or by phone, and take appropriate action to assist him or her.

For patients who are not ready to make a quit attempt, clinicians should use a brief intervention designed to promote the motivation to quit. Specific content areas that should be addressed can be captured by the “5 R’s.” They are:
1. **Relevance.** Encourage the patient to state why quitting is relevant to him or her, being as specific as possible.
2. **Risks.** Ask the patient to identify potential negative consequences of his or her tobacco use, including acute, environmental and long-term risks.
3. **Rewards.** Ask the patient to identify potential benefits, such as improved health, saving money, setting a good example for children and better physical performance.
4. **Roadblocks.** Ask the patient to identify barriers (e.g., fear of withdrawal or weight gain) and provide treatment and resources to address them.
5. **Repetition.** The motivational intervention should be repeated every time the patient is seen.

**Submitting Medicaid Claims for SCC**
Medicaid coverage for smoking cessation counseling is clearly a well-intended adjunct to DOH’s efforts to reduce tobacco use in New York State in which dentists can play an important part. The decision to provide SCC and submit claims for Medicaid reimbursement requires serious consideration by any dentist. To help ensure that reimbursement will be made, it is advisable to:
- Perform all specific requirements enumerated in Medicaid policy.
- Document the time interval, details of the counseling and/or intervention, patient response, follow-up, etc., and adherence to the specific Medicaid requirements.

And, again, dentists who provide treatment to Medicaid recipients enrolled in managed care should review their contracts, confirm with the managed care company how this change will be implemented in writing, and determine whether any change is needed to their participating provider contracts.

*Dr. Shub is NYSDA Assistant Executive Director for Health Affairs.*
Can you tell us something about yourself?

**Where did you grow up?**

I was born in Hong Kong BCC in 1951. I am Chinese by descent. My parents and two older sisters were all born on mainland China. My father was Ping Tchang Liang, M.D. My mother Theresa Chiao-ming Liang. I have two sisters—Hilda and Maria. We were all born about four years apart. My father graduated in 1942 from medical school at Aurora University in Shanghai, a private Catholic university established by the French and run by Jesuit priests. The Jesuits were an extremely strict order and set firm curfews for their students. My father had to sneak out to date my mom. Nonetheless, my parents were extremely devout Catholics throughout their entire life. One of their proudest moments had to have been their visit to the Vatican.

**When did you come to the United States?**

My parents and sisters survived the atrocities of the Japanese invasion and Communist takeover of China. In 1950, my father smuggled our family out of the country through the underground, escaping to Hong Kong. These were extremely dangerous times. My parents lost many close friends to both the Japanese and the Communists. My mother was pregnant with me at the time, and that’s how I was born in Hong Kong, just a ferry ride away from mainland China.

In Hong Kong, my father worked for the British Merchant Marine as a ship’s surgeon and was away for months at a time. My mother cared for the family while he was away. All the while, my father still felt the Communists remained a real threat. It was a near impossible feat, but in 1959, after years of waiting to qualify for a visa, and with a loan from Catholic missions, my father succeeded in bringing our family to the United States. This explains why my father felt indebted to and remained in close touch with Catholic missions throughout his entire life.

My father, in his mid-30s in 1959 when he arrived in the United States, determined to succeed. My family lived in New Jersey for a brief time before settling in Pittsburgh, PA. My father interned at Shadyside Hospital in Pittsburgh, passed his medi-
cal boards and went on to specialize in pathology at St. Joseph’s Hospital, also in Pittsburgh. He was employed at Allegheny Valley Hospital in Natrona Heights, PA, as a pathologist. That’s where he was fondly given his nickname “Ping.” And it was during his tenure there that he proudly became an American citizen.

His becoming a citizen was an indication of how much my parents valued freedom. And it provided an opportunity for all of their children to become educated. My parents’ sacrifices and high regard for education were not wasted. My sister Hilda earned her M.S. in chemistry. She and her husband, Stephen Hui, a retired Ph.D., in biophysics, have relocated to Kennewick, WA, after years of living in Buffalo. My sister Maria received her M.D. from the University of Pittsburgh and went on to specialize in neurology. She and her husband, Mario Ludmer, a retired neurosurgeon, live in North Palm Beach, FL.

My niece, Jennifer, is a neurologist in Monterey, CA, and my nephew, Peter, has his Ph.D. in computer science and is living in Kennewick.

And I have my parents to thank for being where I am today!

Who was most influential in your becoming a dentist?

My high school years were spent growing up in a suburb of Pittsburgh called Natrona Heights. My neighbor across the street, Robert Maple, was a semi-retired dentist. He was a graduate of the School of Dental Medicine at the University of Pittsburgh. He provided me with the most fantastic dental care, personally driving me to and from his small second-story office, showing me how he made his dentures with vulcanite, how he triturated his amalgam, and how he used a dental dam for every procedure (this was in the 1960s). I never knew how meticulous and great a dentist he was until I went to dental school myself years later. He even tried to teach me how to swing a golf club!

Dr. Maple was an important part of my growing up, but my sister Maria was the one who really encouraged me to apply to dental school when the doors of medical school would just not open for me. I have no regrets.

How did you end up in Central New York?

I graduated from the University of Pittsburgh School of Dental Medicine in 1980 and was commissioned as a reserve officer in the Air Force. I was stationed at Griffiss Air Force Base in Rome, in upstate New York. I separated from the Air Force in 1985, having decided to go into private practice. That’s how I ended up in Central New York. I met my wife (since divorced) in the Air Force. Kirsten, my stepdaughter, was 4 years old at the time. In December 1986, son, Kyle, was born—a proud moment in my life. I loved and raised Kirsten as my own. She is now a child life specialist at Boston Children’s Hospital and a graduate of Wheelock College in Boston, where she earned her Master’s degree. She and husband, Brian Getchell, are the proud parents of 2-year-old Connor and 1-year-old Harper.
Kyle graduated from Cortland College with a B.S. in athletic training. He earned his M.S. from Canisius College in Buffalo and is now employed as an athletic trainer at SUNY Cobleskill.

We understand you share your home with several dogs.
True?
My significant other and love of my life, Sharon Tarallo, and I share many things in common. But we both have a strong affection for living things. Before I met Sharon, I had one dog, Biddy, a Westie. Sharon has adopted and rescued many dogs and cats. She has a 7-year-old Australian Shepherd and a cat named Beso (Spanish for “kiss”). Together we saw and fell in love with two eight-week-old sibling rescues from a local foster agency, Dillon and Cassie. The pups were inseparable, so we adopted both of them. The animals get along wonderfully and are extremely affectionate.

What is the last movie you saw?
“Captain Phillips” with Tom Hanks. He is truly an accomplished actor. The movie did not have a dull moment, even though I knew the outcome from the start. One of my favorite TV shows has to be “NCIS.” We also enjoy watching “The Voice.”

What do you do to relax/unwind?
My general dental practice and taking care of four dogs and a cat doesn’t leave me much time for myself. My hobbies include photography, woodworking, home improvements, tinkering with my cars and gardening/landscaping. I also enjoy cooking. My mother taught me a lot of the basics of Chinese cooking, but I also learned by watching the chefs at an upscale Chinese restaurant where I worked as a waiter all through dental school. If I really want to zone out, I park my brain in neutral, get on my big 60” Ferris zero-turn commercial mower and zip around my five acres of lawn on a bright sunny day!

What is your idea of a perfect day?
I would take my entire family, including the grandchildren, to Watkins Glen for the day. We would drive my 2006 Z06 Corvette and my 1990 750iL with a V12 around the famed Watkins Glen racetrack. Then we would stop by the Glenora Winery and have a wonderful lunch out on the deck overlooking the vineyard and Seneca Lake. Of course, the adults would go wine tasting after. My children Kyle and Kirsten actually gave me such a day as a Father’s Day gift about two years ago.

Did you ever imagine you would be president of the New York State Dental Association?
No! The thought never even remotely came to mind in all the years of participating in organized dentistry. It’s like the opportunity presented itself all of a sudden and before I knew it, I was going through the chairs for NYSDA. It is a great honor. I only wish my mother and father could be alive to witness this time in
my life. Sadly, I lost both my parents, my father to acute leukemia and my mother to squamous cell carcinoma.

What are your goals for the coming year?
I look forward to working with my fellow officers, trustees, members of the House and councils, the NYSDA executive director and staff, and grassroots members to continue advancing the wonderful initiatives that have been created by my predecessors, such as recruitment and retention, communications and technology, and fiscal responsibility.

I think the dental profession is hardly immune to economic pressures and political influences. My suspicions were confirmed at the ADA Presidents-Elect Conference in Chicago, and again at this year’s NYSDA Presidents-Elect Conference in Albany. The featured speaker, Marko Vujicic, managing vice president of the ADA Health Policy Institute, spoke to this very issue. The ADA has performed environmental scans and published white papers on the changing dental profession. These changes have already been in effect for some time now. We need to take a hard look at where our profession is headed so that NYSDA and the tripartite can develop strategic plans to help our profession and members continue to succeed in the future.
Root Amputation
A New Look into an Old Procedure


ABSTRACT

A treatment option for managing furcation invasions is root amputation. Long-term survival of resected molars requires a complete harmony of sequential endodontic, periodontic, restorative and maintenance procedures. The main objective of this article is to provide a concise historical perspective of this procedure and to review available literature regarding its efficacy and limitations. It also illustrates a current modification of the procedure using guided bone regeneration (GBR) and socket preservation to eliminate some of the potential disadvantages of the traditional root amputation procedure.

Root amputation—once a common surgical procedure aimed at eliminating furcation invasion in multirooted teeth—was first described by Farrar1 in 1884. It was reintroduced into periodontics by Messinger and Orban in 1954.2 (Similar cases were described in Germany by Gottlieb and Orban in 1933.3) Since then, the procedure has been modified, and its clinical outcomes evaluated longitudinally by several researchers.

The main objective of this article is to provide a historical perspective of root amputation, review available literature regarding its efficacy and illustrate a current modification of the procedure that combines recent advances in bone regeneration and ridge augmentation.

Root Amputation Technique

The indications for root amputation were clearly set forth by Basaraba4 and Staffileno.5 They include Class III furcation involvement; deep Class II furcation; cases of isolated severe bone loss involving one of the roots; vertical root fracture; subgingival root caries; and endodontic indications, such as a persisting periapical pathologic lesion, root resorption or iatrogenic root perforation. Minsk and Polson in 20066 added another indication that includes teeth with high strategic value. This may include teeth with a preexisting fixed prosthesis (as the case presented below) or in cases where anatomic considerations preclude implant placement.

A long-term successful outcome of root amputation depends upon four variables, including meticulous endodontic, periodontic and restorative procedures, and a highly motivated patient.

A prerequisite for root amputation is proper endodontic therapy prior to periodontal surgery in order to determine the feasibility of complete canal obturation. Bühler7 attributed the high
failure rate (32.1%) of root resections to endodontic reasons. It is well documented that multirooted teeth do not respond as favorably as single rooted teeth to endodontic therapy following nonsurgical retreatment or surgical intervention. The complex pulp anatomy of molar teeth presents a greater challenge to eliminating root canal infection, especially when accessory canals are present. The incidence of these canals has been reported to range from 42% to 52% in the mesio-buccal root of maxillary molars.

However, in questionable cases of root proximity or possible root fusion or ankylosis, it is recommended that periodontal surgery be completed prior to endodontic intervention in order to ensure its feasibility. In these cases, vital root amputation could be executed without adverse clinical and histological effects, if the exposed pulp is properly managed. Definitive endodontic therapy should be completed within two weeks to six months, since tooth vitality is significantly reduced six months following a vital root amputation.

A successful root amputation requires complete removal of any ledges or undercuts from the remaining furcation area, after extraction of the resected root. Backman reported four cases of improper or incomplete root resections. In these cases, the roof of the furcation persisted and the sites continued to lose bony support due to an environment that favored biofilm accumulation and retention. To prevent such undue sequelae, it is highly recommended that completeness of the surgical procedure be verified radiographically. Newell examined 70 root-resected teeth, of which 30% were determined to be inadequate due to remaining ledges or residual roots. Majzoub and Kon reported that 86% of resected maxillary first molars exhibited violation of the biologic width. Additionally, only 6% of the examined resected molars had a topography that was easily amenable to periodontal maintenance.

Restoring the remaining tooth properly imposes many clinical challenges. The tooth may require a new fixed prosthesis, which, in turn, may require additional retention with fabrication of a post and core, a procedure that might weaken the root. In order to promote a healthy environment, the new restoration should not violate the biologic width or hinder proper plaque control. Special considerations must be given to the occlusal loads that the tooth has to endure in order to promote longevity. Biomechanical elements and root fractures have been cited as main causes of failure of root amputation. To minimize this risk, the “occlusal table” should be narrow bucco-lingually and free of any occlusal interference.
Because of these factors and the complex nature of this procedure, a suitable candidate must be highly motivated, free of parafunctional habits such as bruxism or risk factors such as smoking. As with any periodontal patient, periodic supportive maintenance treatment is required to prevent recurrence of the disease process.

Long-term survival of root amputation requires a complete harmony of sequential endodontic, periodontic, restorative and maintenance procedures. A shortfall of any step may lead to failure. Review of the literature on its long-term effectiveness has yielded mixed results. Bergenholtz\textsuperscript{20} was first to report on survival of resected molars 11 years postoperatively, with an overall success rate of 85%. Similar success rates were reported by Carnevale and his co-workers\textsuperscript{21} in a retrospective study of resected mandibular molars. These general findings are supported by short- and long-term studies.\textsuperscript{18}

In contrast to these positive results, several studies have reported adverse outcomes and poor long-term survival of resected molars. Langer and his coworkers,\textsuperscript{17} in a retrospective study of 100 resected teeth, reported a high rate of failure of 38% over a 10-year observation period. The main cause of failure in the maxilla was progression of periodontal disease, while mandibular molars succumbed most frequently to root fractures. They concluded that while the initial outcome of resected molars was favorable, it had no lasting effects, and that most failures in their study occurred 5 to 10 years postoperatively.\textsuperscript{17}

In a more recent 10-year retrospective study, Blomlöf and co-workers\textsuperscript{23} evaluated the survival rates of resected molars in 80 patients. The initial survival rate at five years was 89%. This greatly decreased to 68% at 10 years, with smokers demonstrating a greater failure rate than nonsmokers. Dannenwitz and his coworkers\textsuperscript{24} reported a lower success rate of 57.9% for root amputation following an approximately nine-year observation period.

The high failure rate of amputated molars is not surprising considering the complex therapeutic procedures that require the outermost clinical skills and expertise. Furthermore, after extraction of the root(s), resorption of the alveolar bone occurs, resulting in a diminished ridge height and width.\textsuperscript{25} This may lead to permanent deformity of the alveolar ridge and to food impaction under the fixed prosthesis, which complicates optimal oral hygiene. It may also compromise the site for future implant placement, and often causes esthetic problems in patients with an extended buccal corridor.

In light of this negative data and the rise in popularity of dental implants, the procedure has lost its acceptance among many clinicians, while dental implants have been shown to be more predictable with a much higher long-term success rate.\textsuperscript{26}

In order to minimize the aforementioned limitations of the root resection procedure, a modified technique has been used in our clinic with promising results. The principle of guided bone regeneration (GBR) has been employed following the extraction of the resected root(s). Using bone grafts and barrier membranes, the rapidly growing epithelial cells are excluded, allowing the wound to be populated by osteogenic cells.\textsuperscript{27} The literature supports grafting freshly extracted sockets with bone grafts.\textsuperscript{28,29} When freeze-dried bone allograft (FDBA) was used in conjunction with a collagen membrane barrier, the width of the alveolar ridge decreased by only 1.2 mm, compared to a 2.7 mm loss in
untreated, controlled sockets. In addition to width loss, the average loss of bone height in the control group was 1 mm, while the grafted sites gained height. Similar positive results have been reported with other types of allografts and xenografts. The following case illustrates a current technique used in our clinic in conjunction with root amputation.

Case History
A 70-year-old male patient was referred in March 2010 to the graduate periodontal clinic at the University of Tennessee, College of Dentistry, in Memphis due to sharp pain during function on his maxillary left first molar (#14). The patient said the tooth had been treated endodontically twice (in 2006 and 2009) without alleviation of the symptoms. A thorough oral examination revealed the existence of an isolated probing depth of 8 mm and attachment loss on the mesio-palatal root aspect of #14, along with a mesial Class II furcation involvement (Figure 1A). A radiographic survey of the area revealed radiolucency along its mesio-buccal (MB) root (Figure 1B). A fracture of the MB root was highly suspected. The patient declined the extraction option of the offending tooth and replacement with a single dental implant because of his previous negative experience of replacing #15 with an implant. Root amputation of the MB root became a consented viable option.

The occlusion was adjusted to eliminate centric and lateral occlusal contacts on the mesial portion of #14. The area was anesthetized, and buccal and palatal full thickness flaps were reflected from #13 to #14. Removal of the inflamed granulation tissue revealed the presence of a vertical fracture along the MB root and a Class II furcation invasion. The root was amputated using a long diamond bur and high-speed handpiece. Special care was taken to smooth the remaining tooth to remove any ledges or tooth fragments. This was verified clinically and radiographically (Figure 2A).

However, during the extraction process of the offending root, the thin buccal bony wall of the socket was fractured (Figure 2B). Using GBR principles (Figure 3), the area was grafted with a mineralized cancellous particulate allograft (Oragraft, LifeNet Health, Virginia Beach, VA) and covered with an absorbable collagen membrane (ColleTape, Zimmer Dental, Carlsbad, CA). Flaps were repositioned and sutured with vicryl (Ethicon Inc., Johnson & Johnson, New Brunswick, NJ) and chromic gut sutures (Ethicon Inc., Johnson & Johnson, New Brunswick, NJ).

The area healed uneventfully, and the patient did not report any undue consequences. He was placed on a four-month periodontal maintenance recall program. Photographic and radiographic survey of the surgical site at 12 and 27 months (Figures 4 & 5) revealed a healing socket and a preserved bony ridge, along with shallow probing depths.

Summary
This is the first report in periodontic literature that combines the principles of guided bone regeneration in conjunction with an “old procedure” of root amputation. However, a publication by Oh in
2012 in the International Endodontic Journal describes a similar approach to managing periodontic-endodontic lesions with positive results. Further research is needed to verify its long-term efficacy.

Queries about this article can be sent to Dr. Livada at rlivada@uthsc.edu.

REFERENCES


Management of the Dental Patient on Anticoagulant Medication

A Review

Manoj Agarwal, M.D.S.; Sankalp Mittal, M.D.S.; Sharmistha Vijay, M.D.S.; Pooja Yadav, M.D.S.; Vasim Raja Panwar, B.D.S.; Neha Gupta, M.D.S.

A B S T R A C T

Patients taking anticoagulant medication pose a challenge for the clinician. Dentists are often required to manage bleeding as part of routine oral surgery or dental procedures, and altered hemostasis can lead to complications. Nevertheless, use of these medications is generally important for the patient’s health and any alteration in the anticoagulant regimen may have untoward sequelae. In addition, several medications can affect the clotting mechanism, potentially compromising hemostasis. This article will review a variety of anticoagulant medications and the medical conditions that necessitate their use.

Anticoagulation therapy is prescribed for patients when there is a high risk of intravascular clot formation. It may be indicated following embolic stroke, myocardial infarction (MI), atrial fibrillation, placement of a prosthetic heart valve, pulmonary emboli, deep vein thrombosis and any other condition that carries a high risk of intra-arterial or intravenous clotting. Questions commonly arise as to what dental procedures may safely be considered when a patient is on anticoagulant therapy. Generally, controlling bleeding is less of a problem than the management of thrombi and vascular occlusion from decreased coagulopathy.1

Hemostasis depends upon several critical factors. An adequate number of platelets and proper platelet function are essential. The integrity of the vasculature also plays an important role in hemostasis. Vascular integrity can be compromised by vitamin C deficiency, viral and bacterial infections, or as a result of the aging process or through various disease states. Adequate levels of clotting factors and proper functioning of the fibrinolytic pathway are also essential.2

Patient Evaluation and Management

History Taking

A complete medical history should be taken to gather preliminary information about the patient’s general medical condition and should include more detailed questioning about specific medications known or reported to interfere with hemostasis. The specific drug, dose, route of administration and duration of use should be recorded for each medication.

Laboratory Tests

The coagulation cascade involves the intrinsic, extrinsic and common pathways, which have been found to be highly intercon-
nected. The intrinsic pathway requires the clotting factors VII, IX, X, XI and XII. The extrinsic pathway is initiated by the release of tissue factor II. The common point in both pathways is the activation of factor X to factor Xa. Factor Xa activates prothrombin (factor II) to thrombin (factor IIa). Thrombin, in turn, converts fibrinogen to fibrin. Dentists should be able to order and interpret appropriate laboratory tests when minor oral surgery is required for anticoagulated patients. The most frequently used tests are the prothrombin time (PT) and international normalized ratio (INR).

The PT measures the effectiveness of the extrinsic and common pathways. The normal value is approximately 10 to 15 seconds. Because of the variability in PT reported by different laboratories, it is no longer considered adequate to use PT to monitor the level of anticoagulation.

In order to reduce variability, INR was introduced in 1983 by the World Health Organization Committee on Biological Standards to assess patients receiving anticoagulation therapy more accurately. INR is the patient PT divided by the standard PT of the laboratory, raised to the power of the international sensitivity index value (ISI): INR = Patient PT/mean normal PT

A patient with a normal coagulation profile would have an INR of 1.0. It is recommended that a patient undergoing invasive treatment have a PT within 1.5- to 2.0-times the normal value. This corresponds to an INR of 1.5 to 2.5 when the ISI is 1.0.

In patients with anticoagulant therapy, an INR between 2.0 and 3.0 is recommended for most indications. Thus, an INR of 2.5 (range 2.0 to 3.0) minimizes the risk of either hemorrhage or thromboembolism. Conditions such as post-myocardial infarction and deep venous thrombosis (DVT) could cause serious morbidity or death through thromboembolism. Additionally, patients with atrial fibrillation are anticoagulated because of the greater likelihood of blood clot formation due to turbulent blood flow through the heart. In individuals with prosthetic heart valves and certain hypercoagulable states, the INR should be approximately 3.5. If the dentist encounters a patient with an INR greater than 4.0, the patient should be referred to the physician for evaluation. An INR greater than 4.0 is usually considered nontherapeutic, and the patient is at risk for serious bleeding complications.

### Anticoagulant and Antiplatelet Medications

The goal of anticoagulation medication is to prevent clot formation or expansion. Warfarin is the most commonly used anticoagulation drug. Platelets provide the initial hemostatic plug at the site of vascular injury, are involved in pathological processes and are an important contributor to arterial thrombosis leading to MI and ischemic stroke. The most common antiplatelet drugs are acetylsalicylic acid, clopidogrel and dipyridamole.

**Warfarin**

In 1940, Karl Paul Link, a veterinarian from the University of Wisconsin, studied this sweet clover, isolated the active compound and named it WARFARIN, which stands for Wisconsin Alumina Research Foundation. “ARIN” was added to link it to coumarin, because warfarin is a 4-hydroxycoumarin derivative. It is a vitamin K analogue that is rapidly and completely absorbed one hour after ingestion and has a half-life of 36 hours.

Warfarin is also known as coumarin. It was approved for use in humans in 1950 as oral anticoagulant therapy. It is an antagonist of vitamin K, an element necessary for the synthesis of clotting factors II, VII, IX and X, as well as the naturally occurring endogenous anticoagulant proteins C and S. Antagonism of vitamin K or a deficiency of this vitamin reduces the rate at which these factors and proteins are produced, thereby creating a state of anticoagulation.

Warfarin has two functions: anticoagulant activity and antithrombotic effect. Therapeutic doses of warfarin reduce the production of functional vitamin K-dependent clotting factors by approximately 30% to 50% and cause a 10% to 40% decrease in the biologic activity of the clotting factors. As a result, the coagulation system becomes functionally deficient.

**Acetylsalicylic Acid (ASA)**

ASA is an non-steroidal anti-inflammatory drug (NSAID) used to prevent thromboembolic diseases. The antithrombotic action of ASA depends upon the irreversible inhibition of arachidonic cyclo-oxygenase activity in platelets. The recommended dose of ASA is 75 mg to 150 mg daily for the long-term prevention of serious vascular events in high-risk patients.

**Clopidogrel**

Clopidogrel has more antiplatelet activity than ASA and is used to prevent MI and peripheral arterial insufficiency. The antiplatelet effect of clopidogrel is irreversible and lasts for the life of the platelet (7-10 days).

**Dipyridamole**

Dipyridamole inhibits adenosine uptake in erythrocytes and endothelial cells. This increases plasma adenosine levels, leading to more adenosine being available for binding to the adenosine receptor on the platelet. The antiplatelet activity of dipyridamole is less than that of ASA; its action is reversible and ceases about 24 hours after the drug is discontinued.

NSAIDs, other than aspirin—for example, ibuprofen, diclofenac—have a reversible effect on platelet aggregation and platelet...
function, which is restored once the drug is cleared from circulation. NSAIDs are not used clinically for their antiplatelet activity.13

When deciding the appropriate management of a dental patient prescribed warfarin, three questions must be answered: 1. Why is the patient on warfarin? 2. Is it necessary to modify the warfarin regimen? 3. How should the regimen be modified?14

Edward Hui’s recommendations for appropriate management of a patient prescribed oral anticoagulation therapy is shown in Figure 1. The reason for anticoagulation therapy may indicate the need for other precautions.14

**Balancing Bleeding Risks and Thromboembolic Risks**

Patients seen in the dental office may have been instructed by their physician to take one low-dose aspirin daily to prevent thromboembolic events associated with stroke or MI. Cessation of low-dose aspirin therapy prior to oral surgery has been controversial; dentists have been concerned about potential bleeding complications. However, patients who are receiving aspirin therapy are at risk for emboli and MI if the medication is stopped. Studies have shown that patients undergoing minor oral surgery, including implant surgery and third molar extraction, experienced minimal bleeding complications when on low-dose aspirin therapy. These complications were controlled with local measures. The results indicated that low-dose aspirin therapy may be continued even if a patient requires minor oral surgery.

However, many primary care dental procedures are unlikely to cause bleeding that cannot be managed with local measures. These include minimally invasive procedures such as periodontal probing, scaling above the gumline, polishing and orthodontic procedures. Invasive procedures, such as local infiltration, scaling below the gums, root planning, biopsies, tooth extractions, minor periodontal surgery, cavity filling, endodontic procedures (root canals) and prosthodontic procedures (crowns, bridges and implants), are also unlikely to cause significant bleeding. Significant bleeding is more likely to occur with more invasive procedures, such as extraction of impacted teeth and use of periodontal flaps.15-19 Patients with liver disease, kidney disease, hypertension and gingival disease also have an increased risk of bleeding. The skill and experience of the dental practitioner and availability of hemostatic measures must also be considered when deciding whether to stop anticoagulants or antiplatelet agents.20

A frequently cited study of thromboembolic risks in dentistry is Wahl’s literature review, in which serious embolic complications, including death, were three-times more likely to occur in patients whose anticoagulant therapy was interrupted than were bleeding complications in patients whose anticoagulant therapy was continued (and whose anticoagulation levels were within or below therapeutic levels).21

**Maintaining Hemostasis**

With the recommendation not to alter anticoagulant therapy for patients requiring routine dentoalveolar surgery, dentists must adhere to meticulous surgical technique, have the skills to ensure proper wound closure and be familiar with adjunctive hemostatic techniques.

Oral procedures must be done at the beginning of the day, because this allows more time to deal with immediate re-bleeding problems.22

---

**Figure 1. Management of Patient on Oral Anticoagulant (by Edward Hui)14**
Local anesthetic containing a vasoconstrictor should be administered by infiltration or by intraligamentary injection wherever practical.\textsuperscript{2,23} Regional nerve blocks should be avoided when possible. However, if there is no alternative, local anesthetic can be administered cautiously using an aspirating syringe.\textsuperscript{24}

An extraction socket should be gently packed with a resorbable gelatin sponge and then sutured carefully. The gelatin sponge acts as a mechanical matrix to facilitate clotting. Resorbable oxidized cellulose is used similarly to the gelatin sponge. It should be noted that any type of packing material can be a nidus for infection, and caution in the use of these products should be the rule when operating in an infected area. Hemostatic collagen provides a mechanical matrix. When in contact with blood, the collagen causes an aggregation of platelets. This material is also resorbable within 14 to 56 days. Other products that can be used include bone wax, thrombin-soaked gauze and fibrin sealants.\textsuperscript{2,23}

Tranexamic acid 4.8% solution is a topical antifibrinolytic that is commonly used to prevent excessive hemorrhage during surgery. The patient is instructed to rinse with 10 mL of the solution for two minutes, four times a day for seven days.\textsuperscript{2,23}

Resorbable sutures are preferable, as they attract less plaque. If non-resorbable sutures are used, they should be removed after four to seven days. Following closure, pressure should be applied if bleeding continues or restarts. Apply pressure over the socket using a sterile gauze pad for 20 minutes. If bleeding does not stop, the dentist should be contacted.

Look after the initial clot by resting while the local anesthetic wears off and clot fully forms (2-3 hours). Avoid rinsing the mouth for 24 hours. Do not suck hard or disturb the socket with the tongue and any foreign object. Avoid hot liquids and hard foods for the rest of the day. Avoid chewing on affected side until it is clear that a stable clot has formed.

If bleeding continues or restarts, apply pressure over the socket using a sterile gauze pad for 20 minutes. If bleeding does not stop, the dentist should be contacted.

The optimal INR value for dental surgical procedures is 2.5, because it minimizes the risk of either hemorrhage or thromboembolism. Nevertheless, minor dental surgical procedures can safely be done with the INR between 2.0 and 4.0, being aware that local hemostatic measures may be needed to control bleeding. Patients who have an INR greater than 4.0 should not undergo dental surgical procedures, and they must be referred to the clinician responsible for maintaining their anticoagulation.

Conclusion

Most of the recent literature regarding anticoagulants and dental surgery is in agreement that, with good local hemostatic measures, routine oral surgical procedures can be undertaken without the need for alteration of the INR, when it is within the therapeutic range. The management plan is individualized according to the levels of surgical trauma and anticoagulation. Any change of anticoagulation therapy should be made in collaboration with the prescribing physician.

Queries about this article can be sent to Dr. Agarwal at drmnojigdc@gmail.com.

REFERENCES

Angiofibrolipoma of the Retromolar Pad Region
Case Report with a Review of Literature

ABSTRACT

Angiofibrolipoma is a rare histopathological variant of lipoma, characterized by mature adipocytes, blood vessels and dense collagenous tissue. It is seldom seen in the oral and maxillofacial region. Diagnosis of angiofibrolipoma is only possible based on its histopathological features.1 This report presents the case of a 63-year-old male patient with the complaint of a polyp-like mass, felt from the left retromolar pad region. The mass was found as a small prominent lesion that had grown gradually for 1.5 years. Our differential diagnosis was irritation fibroma and fibrous pyogenic granuloma. This report also includes a comprehensive reference to previously reported data, as found through PubMed and Google Search, which revealed this type of case rarely has been documented.

Lipomas present benign mesenchymal tumors composed of mature adipocytes. They are well separated from surrounding tissues by a thin fibrous capsule.1-3 Although lipomas are among the most common mesenchymal neoplasms, the head and neck regions are affected in 15% to 20% of cases, and only 1% to 4% of these tumors are located in the oral cavity.3-5 The most affected anatomical sites in the oral cavity are the buccal mucosa, lips and tongue. The floor of the mouth, palate, retromolar pad and salivary glands are involved less frequently.3

Lipomas are lesions of the middle-aged, seen mostly in patients older than 40 years.1 They seldom present before the third decade of life; and they have a slight gender predilection towards males.6 However, some studies have shown a female preponderance, while others did not find any gender preference.7

Clinically, oral lipomas are slow-growing tumors in the form of well-circumscribed, mobile, painless, sub-mucosal, sometimes fluctuant, yellowish-colored nodules, depending upon the thickness of the overlying mucosa.1,6,5

There are two alternative classifications for lipomas, which is important for differential diagnosis. WHO’s classification consists of classic lipoma, angiolipoma, chondroid lipoma, myolipoma and spindle cell/pleomorphic lipoma, according to their characteristic clinical and histopathological features.8,9 In the alternative classification, histological variants of lipomas include fibrolipomas, angiolipomas, angiofibrolipomas, angiomyolipomas and infiltrating angiolipomas.10,11

The first description of an oral lesion was provided in 1848 by Roux in a review of masses, which were referred to as a “yellow epulis.” The pathogenesis of lipoma is uncertain, but it appears to
be more common in obese people. However, the metabolism of lipoma is completely independent of normal body fat. If the caloric intake is reduced, normal body fat may be lost, but lipomas do not decrease in size. Although its etiology is unknown, possible causes can include trauma, infection, and chronic irritation and hormone alterations.4

In this paper, we report one case of angiofibrolipoma in the left retromolar pad region of a 63-year-old man. We believe this is the first reported case of angiofibrolipoma located in this area in a male patient.

Additionally, we have searched for previous case reports and carried out an analysis of the data on the different histological types of intraoral lipomas, which we present in this paper.

Case Report
A 63-year-old male was referred to the Department of Oral Medicine, School of Dentistry, Tehran University of Medical Science, Iran, with the complaint of a polyp-like mass, felt from the left retromolar pad. From the past history, it was discovered that the mass appeared 1.5 years previously as a small prominent area on the retromolar pad and grew gradually.

Intraoral examination revealed a polypoid mass, about 1 cm in diameter, located on the left retromolar pad. The color of the covering mucosa was the same as the normal surrounding mu-
<table>
<thead>
<tr>
<th>Author</th>
<th>Num</th>
<th>Year</th>
<th>Age</th>
<th>Gender</th>
<th>Location</th>
<th>Histological Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brucoli</td>
<td>1</td>
<td>2011</td>
<td>43</td>
<td>M</td>
<td>Right buccal mucosa</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Kaur</td>
<td>3</td>
<td>2011</td>
<td>54</td>
<td>M</td>
<td>Labial mucosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>52</td>
<td>M</td>
<td>Buccal mucosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>55</td>
<td>M</td>
<td>Buccal mucosa</td>
<td></td>
</tr>
<tr>
<td>Oliveira dos santos</td>
<td>1</td>
<td>2011</td>
<td>58</td>
<td>M</td>
<td>Right jugal mucosa above alveolar ridge and fromen mentonian</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Taira</td>
<td>1</td>
<td>2011</td>
<td>65</td>
<td>F</td>
<td>Extending superiorly from mandibular gingivobuccal fold to gingiva</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Venkateswarlu</td>
<td>1</td>
<td>2011</td>
<td>6</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Rafieyan</td>
<td>1</td>
<td>2011</td>
<td>60</td>
<td>M</td>
<td>Tongue</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Martinez-Mata</td>
<td>1</td>
<td>2011</td>
<td>12</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Omo</td>
<td>1</td>
<td>2011</td>
<td>52</td>
<td>M</td>
<td>Tongue</td>
<td>Myxolipoma</td>
</tr>
<tr>
<td>Caldeira</td>
<td>2</td>
<td>2011</td>
<td>38,60</td>
<td>M,M</td>
<td>Buccal mucosa, hard palate</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Nayak</td>
<td>1</td>
<td>2011</td>
<td>63</td>
<td>M</td>
<td>Right palatal</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Amirth Raj</td>
<td>1</td>
<td>2011</td>
<td>72</td>
<td>M</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Joswal</td>
<td>1</td>
<td>2011</td>
<td>65</td>
<td>F</td>
<td>Buccal mucosa</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Gupta</td>
<td>1</td>
<td>2011</td>
<td>2,5</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Brnicic</td>
<td>1</td>
<td>2010</td>
<td>59</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiofibrolipoma</td>
</tr>
<tr>
<td>Thakur</td>
<td>1</td>
<td>2010</td>
<td>8</td>
<td>F</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Majunatha</td>
<td>3</td>
<td>2010</td>
<td>Mean:66.6</td>
<td>M</td>
<td>2 Buccal mucosa, 1 palate</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Rehani</td>
<td>1</td>
<td>2010</td>
<td>42</td>
<td>F</td>
<td>Buccal mucosa</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Altug</td>
<td>1</td>
<td>2009</td>
<td>22</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Colella</td>
<td>1</td>
<td>2009</td>
<td>75</td>
<td>M</td>
<td>Tongue</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Annibali</td>
<td>1</td>
<td>2009</td>
<td>58</td>
<td>F</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Ayberk Alug</td>
<td>1</td>
<td>2009</td>
<td>22</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Adoga</td>
<td>1</td>
<td>2008</td>
<td>35</td>
<td>F</td>
<td>Buccal mucosa</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Scarlott</td>
<td>1</td>
<td>2008</td>
<td>71</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Imai</td>
<td>1</td>
<td>2008</td>
<td>72</td>
<td>M</td>
<td>Bilateral margins of tongue</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Brooks</td>
<td>3</td>
<td>2008</td>
<td>8</td>
<td>F</td>
<td>2 Buccal mucosa, 1 mandibular labial/buccal vestibule</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Kacker</td>
<td>1</td>
<td>2007</td>
<td>78</td>
<td>M</td>
<td>Tongue</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Coimbra</td>
<td>1</td>
<td>2006</td>
<td>29</td>
<td>F</td>
<td>Laterally to opening of right duct of Wharton</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Chizonda</td>
<td>1</td>
<td>2006</td>
<td>58</td>
<td>F</td>
<td>Tongue</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Tosios</td>
<td>1</td>
<td>2006</td>
<td>55</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Wetzner</td>
<td>1</td>
<td>2005</td>
<td>4</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Ghandour</td>
<td>1</td>
<td>2005</td>
<td>72</td>
<td>M</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Dale</td>
<td>1</td>
<td>2005</td>
<td>8</td>
<td>F</td>
<td>Soft palate</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Yonemochi</td>
<td>1</td>
<td>2005</td>
<td>69</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Metgud</td>
<td>1</td>
<td>2004</td>
<td>17</td>
<td>M</td>
<td>Tongue</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Vera 4</td>
<td>1</td>
<td>2004</td>
<td>74</td>
<td>M</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Tan</td>
<td>1</td>
<td>2004</td>
<td>55</td>
<td>M</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Epivatianos</td>
<td>1</td>
<td>2003</td>
<td>23</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Myxolipoma</td>
</tr>
<tr>
<td>Kaku</td>
<td>1</td>
<td>2003</td>
<td>75</td>
<td>M</td>
<td>Bilateral margins of tongue</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Agolf</td>
<td>1</td>
<td>2001</td>
<td>61</td>
<td>F</td>
<td>Right gingivobuccal sulcus</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Pattelli</td>
<td>1</td>
<td>2000</td>
<td>6</td>
<td>F</td>
<td>?</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Dutt</td>
<td>1</td>
<td>1999</td>
<td>42</td>
<td>F</td>
<td>?</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Aghshari</td>
<td>1</td>
<td>1997</td>
<td>3</td>
<td>F</td>
<td>?</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Dattilo</td>
<td>1</td>
<td>1996</td>
<td>3</td>
<td>F</td>
<td>?</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Khoa</td>
<td>1</td>
<td>1995</td>
<td>23</td>
<td>M</td>
<td>Buccal/labial mucosa</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Anavi</td>
<td>1</td>
<td>1995</td>
<td>70</td>
<td>M</td>
<td>Floor of mouth</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Lombardi</td>
<td>1</td>
<td>1994</td>
<td>68</td>
<td>F</td>
<td>Tongue</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Begin</td>
<td>1</td>
<td>1993</td>
<td>42</td>
<td>F</td>
<td>Left palatine tonsil</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Christopoulos</td>
<td>1</td>
<td>1989</td>
<td>58</td>
<td>M</td>
<td>Hard palate</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Krauser</td>
<td>1</td>
<td>1986</td>
<td>3</td>
<td>F</td>
<td>Tonsil</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Vazirani</td>
<td>1</td>
<td>1985</td>
<td>3</td>
<td>F</td>
<td>?</td>
<td>Fibrolipoma</td>
</tr>
<tr>
<td>Tripp</td>
<td>1</td>
<td>1985</td>
<td>3</td>
<td>M</td>
<td>Buccal vestibule</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Mcdaniel</td>
<td>1</td>
<td>1984</td>
<td>33</td>
<td>F</td>
<td>Anterior floor of mouth</td>
<td>Spindle cell lipoma</td>
</tr>
<tr>
<td>Campos</td>
<td>1</td>
<td>1980</td>
<td>44</td>
<td>M</td>
<td>Buccal mucosa</td>
<td>Angiomyxolipoma</td>
</tr>
<tr>
<td>Shapiro</td>
<td>2</td>
<td>1969</td>
<td>5</td>
<td>M</td>
<td>?</td>
<td>Lipoma</td>
</tr>
<tr>
<td>Orlean</td>
<td>1</td>
<td>1961</td>
<td>5</td>
<td>M</td>
<td>Labial mucosa</td>
<td>Lipoma</td>
</tr>
</tbody>
</table>

M: male  F: female  ?: Data were not accessible
Lipomas are similar to normal fat tissue. But their metabolism differs, because these lipids are not available for normal metabolism. Lipomas are similar to normal fat tissue. But their metabolism is quite different, because these lipids are not available for normal metabolism. The etiology of lipoma is still unknown. It varies from the differentiation of multi-potent mesenchymal cells in fat tissue, cartilage and bone, to metaplasia of a pre-existing lipoma. Mesenchymal cells are modified by systemic and local influences that range from local trauma to prolonged ischemia. Further indistinct etiology of osseous/chondroid change in lipoma has been discussed; and most researchers have mentioned that their origin is from different types of undifferentiated mesenchymal cells.

Piattelli et al. described two hypotheses for the origin of chondroblasts and osteoblasts. One hypothesis is that the neoplastic transformation occurs in multi-potent, undifferentiated mesenchymal cells that later differentiate into lipoblasts, chondroblasts, or osteoblasts and fibroblasts. The other hypothesis is that only the adipose cells have a neoplastic transformation, and the cartilage and bone are produced by differentiation of undifferentiated mesenchymal cells of stroma in chondroblasts or osteoblasts. Various pathogenic mechanisms, such as an origin from lipoblastic embryonic cell nests, metaplasia of muscle cells and fatty degeneration, have been proposed as putative causative factors for simple lipoma. Other factors, such as trauma, infection, chronic infection and hormonal imbalance, are also thought to have a role. Additionally, it is believed that diabetes mellitus induced by hypercholesterolemia and obesity, radiation, and a familial or genetic link, such as abnormality of chromosomes, may be involved in lipoma development. Few lipomas show rearrangement of 12q, 13q or 6p chromosomes. They usually have chromosomal aberrations, such as translocations involving 12q13-15, locus interstitial deletions of 13q, and rearrangements involving 8q11-13 locus.

However, in the literature, trauma is also mentioned as one of the etiological factors. There are two different opinions about the occurrence of so-called “traumatic lipomas.” The first is that after trauma, adipose tissue prolapsed through fascia results in a direct impaction. The second opinion is that after soft-tissue trauma and hematoma formation, cytokines mediate differentiation and proliferation of pre-adipocytes, resulting in lipoma formation. While most lesions are developmental anomalies, those that occur in the maxillofacial region usually arise late in life and are presumed to be neoplasms of adipocytes, occasionally associated with trauma. Oral lipomas located on the buccal mucosa may not represent true tumors but, rather, herniation of the buccal fat pad through the buccinator muscle. Such cases may occur subsequent to local trauma in young children or the surgical removal of third molars in older patients.

Among the rarest seen histopathological variants of lipoma is angiofibrolipoma. This neoplasm is composed of fibroblasts, capillaries and adipose tissue that is not encapsulated, but is well separated from neighboring tissues. In the literature, there are few reports of angiofibrolipoma associated with the oral and maxillofacial region. Brick et al. reported a case of angiofibrolipoma of the buccal mucosa in a 59-year-old female. However, our case was reported in a male in the same age group, and the lesion was located on the retromolar pad region. A report by Jacob et al. described a case of ear canal angiofibrolipoma. Also, Krause et al. reported a tonsillar angiofibrolipoma.

**Discussion**

Intraoral lipomas are relatively rare tumors, although they occur with higher frequencies in other areas, especially the back, abdomen and shoulder of adults. Lipomas of the oral cavity are diagnosed more frequently in adult patients at a mean age of 50.2 to 62 years. These data are consistent with the 63-year-old subject of the study presented here. In contrast to the male predominance of lipomas involving other regions of the body, in the case of lipomas of the oral cavity, studies have demonstrated a relatively balanced gender distribution, or a slight bias toward women. However, in this case, the report concerns a male patient. An important characteristic of oral lipomas is their small size, with a diameter of 1 cm to 3 cm. In this case, the mass was 1 cm in diameter.

Lipomas are similar to normal fat tissue. But their metabolism is quite different, because these lipids are not available for normal metabolism. The etiology of lipoma is still unknown. It varies from the differentiation of multi-potent mesenchymal cells in fat tissue, cartilage and bone, to metaplasia of a pre-existing lipoma. Mesenchymal cells are modified by systemic and local influences that range from local trauma to prolonged ischemia. Further indistinct etiology of osseous/chondroid change in lipoma has been discussed; and most researchers have mentioned that their origin is from different types of undifferentiated mesenchymal cells.

Piattelli et al. described two hypotheses for the origin of chondroblasts and osteoblasts. One hypothesis is that the neoplastic transformation occurs in multi-potent, undifferentiated mesenchymal cells that later differentiate into lipoblasts, chondroblasts, or osteoblasts and fibroblasts. The other hypothesis is that only the adipose cells have a neoplastic transformation, and the cartilage and bone are produced by differentiation of undifferentiated mesenchymal cells of stroma in chondroblasts or osteoblasts. Various pathogenic mechanisms, such as an origin from lipoblastic embryonic cell nests, metaplasia of muscle cells and fatty degeneration, have been proposed as putative causative factors for simple lipoma. Other factors, such as trauma, infection, chronic infection and hormonal imbalance, are also thought to have a role. Additionally, it is believed that diabetes mellitus induced by hypercholesterolemia and obesity, radiation, and a familial or genetic link, such as abnormality of chromosomes, may be involved in lipoma development. Few lipomas show rearrangement of 12q, 13q or 6p chromosomes. They usually have chromosomal aberrations, such as translocations involving 12q13-15, locus interstitial deletions of 13q, and rearrangements involving 8q11-13 locus.

However, in the literature, trauma is also mentioned as one of the etiological factors. There are two different opinions about the occurrence of so-called “traumatic lipomas.” The first is that after trauma, adipose tissue prolapsed through fascia results in a direct impaction. The second opinion is that after soft-tissue trauma and hematoma formation, cytokines mediate differentiation and proliferation of pre-adipocytes, resulting in lipoma formation. While most lesions are developmental anomalies, those that occur in the maxillofacial region usually arise late in life and are presumed to be neoplasms of adipocytes, occasionally associated with trauma. Oral lipomas located on the buccal mucosa may not represent true tumors but, rather, herniation of the buccal fat pad through the buccinator muscle. Such cases may occur subsequent to local trauma in young children or the surgical removal of third molars in older patients.

Among the rarest seen histopathological variants of lipoma is angiofibrolipoma. This neoplasm is composed of fibroblasts, capillaries and adipose tissue that is not encapsulated, but is well separated from neighboring tissues. In the literature, there are few reports of angiofibrolipoma associated with the oral and maxillofacial region. Brick et al. reported a case of angiofibrolipoma of the buccal mucosa in a 59-year-old female. However, our case was reported in a male in the same age group, and the lesion was located on the retromolar pad region. A report by Jacob et al. described a case of ear canal angiofibrolipoma. Also, Krause et al. reported a tonsillar angiofibrolipoma. Saddik et al. reported...
a case of lip sarcoma of the base of the tongue and tonsillar fossa in a patient who underwent several resections of the mass, which was once diagnosed as angiofibrolipoma.\textsuperscript{19} We believe this is the first reported case of angiofibrolipoma located on the retromolar pad region in a male patient. Histopathological analysis of the biopsy specimen taken from the retromolar pad revealed numerous vascular channels surrounded by collagen-rich fibrous tissue and mature adipocytes, which was diagnosed as angiofibrolipoma.

The clinical differential diagnosis may include ranula, dermoid cyst or thyroglossal duct cyst, regardless of its location and ectopic thyroid tissue, pleomorphic adenoma and mucoepidermoid carcinoma, angiolipoma, fibrolipoma, granular cell tumor, neurofibroma, traumatic fibroma and malignant lymphoma. The definitive diagnosis is made by means of microscopic examination, which shows adult fat tissue cells embedded in a stroma of connective tissue and surrounded by a fibrous capsule.\textsuperscript{6,7,20}

Surgical excision of lipomas is the preferred treatment modality, and if adequately resected, recurrence is rare. Therefore, complete resection should be emphasized during the first surgical operation. Recurrence is more likely in deep lipomas, which have a recurrence rate of 30\% to 50\%, probably because of the difficulty of complete surgical removal.\textsuperscript{1,7,14} Well-encapsulated lipomas easily shell out with no possibility of recurrence or damage to the surrounding structures.\textsuperscript{7} The surgical approach is dependent upon the site of the tumor and the proposed cosmetic result. Lesions outside of the oral cavity may show greater recurrence rates after surgical excision.\textsuperscript{5}

Conclusion

This paper reports the case of a left retromolar pad angiofibrolipoma in a 63-year-old male. In referencing the literature to date, this type of case has been rarely documented. 

The authors thank Dr. K. Drudgar and Dr. M. Mirza-Mohammad in the Department of Oral Medicine and their colleagues in oral pathology in the Dentistry School, Tehran University of Medical Sciences, Tehran, Iran, for their assistance in the preparation of this report. Queries about this article can be sent to Dr. Agha-Hosseini at aghahose@sina.tums.ac.ir.

REFERENCES

The Residual Radicular Cyst


ABSTRACT

The existence of a true residual radicular cyst has been called into question. When observed, it probably represents, with rare exceptions, a resolving radicular cyst—a "work in progress."

The epithelial debris of Malassez represents the remnants of the sheath of Hertwig. The debris resides in the tooth’s periodontal ligament. With tooth devitalization, the apically located epithelial debris becomes incorporated into the developing periapical granuloma. Bacteria, present in the necrotic pulp, act as the antigenic stimulus for the formation of the granuloma. In turn, cytokines, proteins secreted by the cells present in the granuloma, stimulate proliferation of the dormant periodontal epithelial debris.1-4

Eventually, the central mass of the proliferating epithelium becomes deprived of its vascular nutrition and degenerates to form a cyst cavity. With shedding of epithelial cells and lytic products into the cavity, an increase in osmotic pressure occurs.2,5 The resulting fluid intake increases the cyst lumen’s hydrostatic pressure and leads to bone resorption as the cyst expands.1 Alternatively, it has also been suggested that proliferating epithelium will surround any apical abscess initiated by an infected necrotic pulp. Because of its innate nature, the epithelium will proliferate to cover the exposed connective tissue surfaces and form an epithelially lined cyst cavity.2

The seminal microscopic review by Bhaskar6 of 2,308 periapical lesions indicated that 48% were granulomas, while 42% of the lesions were radicular cysts. Subsequent studies reported that a varying incidence of 6% to 55% of the periapical lesions were cysts.2,7-9 With the known, very high success rates following endodontic therapy, it is apparent that routine endodontic care will eradicate these cysts.5,6,8,10,11 Many periapical lesions are left behind following dental extractions. And because many are radicular cysts, their presence should frequently be recognized postoperatively years later. Nevertheless, the residual radicular cyst (RRC) has proven to be a rarity. Therefore, it would seem that resolution of a periapical radicular cyst can also occur following removal of the causative tooth.10-12

The relatively rare RRC is best defined as an odontogenic cyst that persists after the associated tooth has been extracted.13 It is generally believed that the majority of them represent slowly resolving radicular cysts.10,12 Although most RRC resolve with removal of the irritant (the tooth), a small percentage persist for unknown reasons.12 Inflammation from some form of irritation may be the key factor in the continued presence or growth of the RRC. Failure to eliminate the irritant, or its reactivation, can serve to maintain the epithelial lining or even activate its further prolif-

From the Department of Oral and Maxillofacial Surgery, Columbia University College of Dental Medicine, New York, NY
eration. However, it can be expected that the RRC will resolve if the associated inflammatory process also recedes.

Clinically, the RRC is recognized as a well-defined, asymptomatic, circumscribed radiolucency in an edentulous area. It is reported to occur more commonly in the anterior maxilla, usually in males whose ages range from 30 to 60 years. Radiologically, the RRC has a well-defined periphery of cortical bone. Its size and lucency will decrease with the passage of time. Histologically, there is a thinning of the cyst's differentiated epithelial lining and a decrease in the cyst wall’s inflammatory cells. These cells reflect the granuloma that pre-existed the original radicular cyst. A mature avascular connective tissue wall surrounds the cyst.

Case Report

A 44-year-old African-American male was seen in Columbia University College of Dental Medicine for a routine dental examination. When the radiologic examination revealed a well-defined lucency in the area of the missing maxillary right lateral incisor (Figure 1), the patient was referred to the oral and maxillofacial surgery service.

A history indicated that the patient was in excellent health. He denied all systemic diseases; and he stated he was using no medications. A non-vital maxillary right lateral incisor had been extracted approximately 10 months previous to his present visit. Healing had been uneventful; and no subjective discomfort or swelling has developed. Because he is aware that he has other carious teeth, he now seeks further care.

Extraorally, no anterior facial swelling was apparent. Intraorally, a normally healed mucosa overlying the edentulous maxillary right lateral incisor area was observed (Figure 2). The adjoining mucobuccal fold was not swollen, and palpation of the entire region around the edentulous area elicited no discomfort. Carious involvements of the adjacent vital canine and central incisor were present.

A radiograph of the involved area revealed that a well-defined circular lucency, measuring approximately 1 cm in diameter, occupied the alveolar process in the right maxillary lateral incisor region. No connection of the lucency to the adjacent canine or central incisor was noted. The lucency was delineated by a layer of cortical bone.

A decision was made to intervene surgically. A mucoperiosteal flap was mobilized exposing an intact buccal alveolar plate. A surgical bur was used to expose an encapsulated soft tissue mass, which was readily enucleated from its bony crypt (Figure 3). After debridement, the flap was reflected and coapted with sutures. Healing was rapid. Two weeks later, the patient was referred for restorative dental care.

The histologic diagnosis made by the oral pathologist was RRC. A lumen surrounded by a stratified squamous epithelial lin-
ing based on a relatively non-flamed connective tissue wall was identified (Figure 4). Rushton bodies were noted.

Discussion
There are only rare and sporadic reports of the RRC. Because our case fits the criteria for the diagnosis of the RRC, the authors wish to call the profession’s attention to the uncommon existence of this cyst. It can be presumed that in our patient the previously extracted devitalized lateral incisor was associated with a radicular cyst. Resolution of the cyst could be expected because the irritant (the tooth) was removed. Probably the resolution was ongoing, and the process had not yet been completed in the 10-month time frame between extraction of the lateral incisor and the patient’s visit to our clinic. It is also possible that although most RRCs resolve with time, a small percentage is static in behavior and persists unchanged in configuration.12

Another reason for the presence of the RRC is failure of the pre-existing inflammation to resolve following extraction or endodontic care. Additionally, the reinstitution of a new inflammatory irritant that serves to stimulate further epithelial proliferation may occur. Inhibition of resolution from inflammatory stimulation does not seem to be the problem in our reported case because histologically, inflammation was not deemed to be significant (Figure 4). Rather, we believe that resolution of the pre-existing radicular cyst was proceeding as a “work in progress.” We intervened surgically before the process had a chance to be completed. The absence of an inflammatory infiltrate in the cyst wall and a well-defined periphery of cortical bone suggest such a conclusion. Rushton bodies, identified during the histologic examination, occur in about 8% of RRCs.15 These bodies are thought to represent eosinophilic straight, rounded or curved irregular structures with an epithelial lining.15 They probably develop when secretory products of odontogenic epithelium are deposited on particulate matter, such as cell debris or cholesterol crystals, in the cyst wall.15

Conclusion
The existence of the RRC as a separate pathologic entity has been questioned. It is best interpreted as an intermediate healing stage of a radicular cyst following removal of the cyst’s etiologic cause. The rare exceptions that persist probably result from failure to eradicate the inflammatory condition that may exist in the cyst’s wall or if a new inflammatory state is initiated by an exogenous cause. 

Queries about this article can be sent to Dr. Mandel at LM7@Columbia.edu.

REFERENCES
Treatment of a Mandibular Cyst Before Implant Placement

Case Report

Sachin Mamidwar, M.B.B.S, M.S.

ABSTRACT

The aim of this case study is to present a clinical approach to treatment of a mandibular intrabony cyst employing guided bone regeneration principles and protection of the mandibular nerve prior to implant placement. A treatment approach employing a combination of grafting materials and membranes was used to treat the cyst and protect the mandibular nerve prior to implant placement. Micro CT, as well as histology and histomorphometrics, was used to evaluate treatment outcomes. Histological inspection showed bone regeneration at the grafting site. Histomorphometric analysis of the biopsy core rendered a total bone percent area of 58.87% and 41.13% soft tissue. Out of the total bone percent area, 90.45% was revealed as vital bone and 9.55% was graft remnant. The grafted area is supporting an implant-supported prosthesis in full function.

Human bone has the capacity to heal itself through regeneration following different pathologies, such as fractures and resections. Odontogenic cysts are a common occurrence in the dental surgical field. The standard care of treatment, as described by Partsch, for such pathologies is surgical enucleation of the cyst, followed by suturing (Partsch 1910). There has been some debate about the size of the cysts that can be treated through cystectomy. Partsch said the limit should be 2 cm, otherwise complications would arise. However, literature studies reported positive results when different size cystectomy defects were treated in the conventional way without any adjuvant grafting material.1-3 In 1965, Schulte introduced a collagen sponge into the cystectomy defects, based on the notion that the blood clot cannot stabilize in a large defect. Since then, numerous materials have been investigated as possible treatments for large bone defects. Autografts, allografts, xenografts and alloplasts have been widely investigated. The “gold standard” of treatments has been autografts; however, cystic defects are large and autologus bone is scarce.

Calcium sulfate (CS) has often been used in combination with other graft materials, such as allografts. Mixing CS with particulates of different bone graft materials helps to bind the particulates together and prevents the composite graft from migrating out of the site.4-8 Also, this mixture aids in the handling and insertion of particle-based grafts. It was reported by Sottosanti et al. that the combination of CS and allograft results in better bone formation compared to allograft alone. In this study, a combination of CS and demineralized freeze-dried bone allograft (DFDBA) was used in the treatment of periodontal defects.9 The researchers found that CS binds the graft particles and accelerates the mineralization process, thus enhancing bone regeneration.10
Methods and Materials
A patient presented in private practice for implant treatment. Following medical and dental history, the patient reported removal of an intrabony cyst. This cyst was treated by a previous clinician with extraction of two posterior teeth and a marsupialization procedure. The biopsy report obtained from the previously treating clinician stated: “Large intrabony destructive lesion left mandible which involves multiple teeth and extends significantly inferior. Clinical diagnosis: R/O neoplasia vs. possible inflammatory cystic lesion. Final diagnosis: Consistent with inflamed cyst of mandible.”

After waiting 14 months from the previous marsupialization procedure, cone beam computed tomography and re-entry to lower left mandibular quadrant confirmed recurrence of a large boney cyst. A full thickness flap was raised, and the site was completely curetted and debrided of all granulation tissue. Upon removal of tissue, all remaining mandibular walls were intact, with a large defect resulting within the confines of these boney walls (Figure 1). The defect was in close approximation to the ceiling of the mandibular canal.

In order to protect the mandibular canal, a Helios collagen membrane was placed over the mandibular canal (Figures 2, 3). A mixture of calcium sulfate (DentoGen, Orthogen LLC, Springfield, NJ) and allograft Puros small particle, 250 micron to 1,000 micron, (Zimmer Dental, Carlsbad, CA) in a ratio of 40% to 60% CS:P was applied. Collatape was placed over the entrance to the cystic cavity.
The soft tissues were repositioned using a resorbable vicryl suture (Ethicon, Inc., Somerville, NJ) (Figure 5).

The sutures were removed seven days later. The graft was left for 11 months to heal until implant placement (Figure 6). Only after drilling past the crestal bone cortex was a trephine drill used to obtain a core, thus eliminating any pre-existing bone and only obtaining bone in the grafted site (Figure 7). The core of the osteotomy was submitted for micro CT analysis, as well as histological and histomorphometric analysis (Figures 10-13).

**Histological Protocol**

Retrieved bone core was fixed in 10% phosphate buffered formalin and then transferred to different gradients of alcohol concentrations (70% ethanol for 24 hours, 95% ethanol for 24 hours, 100% ethanol [x2] for 48 hours). After dehydration, the sample was infiltrated and embedded in polymethyl methacrylate (PMMA). Sectioning was performed with a low-speed saw (Isomet, Buehler, Lake Bluff, IL). The sample was glued on a Plexiglas slide and ground down and polished to a thickness of approximately 100 μm. The stain used was Stevenel’s Blue and Van Gieson’s Picro-Fuchsin. A slide scanner (ScanScope GL, Aperio, Vista, CA) was used to image the slide and histomorphometrical analysis, using Leica QWin software. It was conducted to quantify the amount of total vital bone present in the core. Since the human allograft can stain similar to vital bone, the presence of stained cells was a positive indicator for vital bone.

**Results**

The patient’s healing occurred without complications. The grafted area presented keratinized soft tissue, without signs of inflammation or infection. Following flap elevation, inspection and probing revealed robust bone formation, with a suitable ridge that allowed for proper placement of an implant based on a surgical guide (Figures 8, 9).

Histological inspection showed bone regeneration at the grafting site (Figure 10). Histomorphometric analysis of the biopsy core rendered a total bone percent area of 58.87% and 41.13% soft tissue. Out of the total bone percent area, 90.45% was revealed as vital bone and 9.55% as graft remnant.

**Discussion**

Treatment modalities to surgically remove cysts include enucleation, marsupialization, curettage, removal of the content with curettage, or, occasionally, surgical resection (1,11-13). After removal of the cyst, guided bone regeneration principles facilitate osteoblasts and other osteogenic factors to occupy and play a critical role in the healing and regenerative phases in a boney defect. Although some clinicians may wait less time to allow for healing before implant placement, it was decided, based on the size of the intrabony defect, to wait 11 months.

During osteotomy preparation for implant placement, a dense bone was encountered in the grafted area. This clinical impression
was supported by the histology and histomorphometric analysis, which showed 58.87% total bone present in the core, of which 90.45% is newly generated bone. The patient received a fixed screw-retained implant-supported prosthesis that is both functional and comfortable. The placement of a Helios membrane was for two purposes: 1. to help protect the mandibular canal; and 2. to establish a base on which to place the bone graft inferiorly. By employing both an allograft and calcium sulfate, a scaffold was established that aided in osteoconduction and aided in the osteogenic cascade during the healing phase. One proposed mechanism of calcium sulfate biochemistry is that it can break down into calcium and sulfate ions, thereby leaving free calcium ions to bind with phosphate, which will allow for bone deposition and remodeling.

In addition, calcium sulfate lowers the pH and helps nearby bone release bone morphogenic proteins like BMP2. Clinically, implants have successfully osseointegrated in the grafted area and are supporting a screw-retained prosthesis (Figure 9).

Conclusion
Following curettage, the combination of an allograft and calcium sulfate employing principles of guided bone regeneration resulted in new bone fill and functional regeneration for a patient who presented with a large boney defect replacing two posterior teeth.

Queries about this article can be sent to Dr. Yacker at drmyacker@yackerdental.com.

REFERENCES
Oral and Dental Manifestations of Celiac Disease


A B S T R A C T

Celiac disease is an autoimmune disease characterized by the malabsorption of nutrients because the villi of the small intestines are unable to process these nutrients. It is brought on by gluten food products. A pattern of enamel defects and oral aphthae are common findings in celiac disease, thus making the dentist an integral part of the diagnostic team.

The dentist plays a vital role in the diagnosis of celiac disease (CD), as it manifests itself dentally with bilateral enamel defects that follow the chronological pattern of tooth development. Also, oral aphthae are commonly seen in patients with CD. The dentist has the ability to assimilate the information gathered from the dental examination, the patient’s medical history and the intraoral examination and formulate a tentative diagnosis of CD. The dentist would then refer the patient to the proper medical specialist for appropriate diagnostic tests and counseling. This would be a valuable service, as the diagnosis of CD can be difficult and is often misdiagnosed, leading to much suffering and worry on the part of the individual with undiagnosed disease.

Celiac Disease

CD is a chronic systemic autoimmune disease that occurs in genetically susceptible individuals. It was first recognized in 1887. Its prevalence is between 1% and 2% of the population in some nations. There are a number of gastrointestinal manifestations of this disorder, such as diarrhea, weight loss, abdominal pain and irritability. A thorough list of the systemic manifestations of the disorder is presented in Table 1. Some individuals with CD do not have diarrhea or weight loss but, rather, have iron deficiencies or alterations in blood chemistry.

CD damages the villi of the small intestine. The villi act to absorb vitamins, minerals and various other nutrients. In CD, they are damaged, resulting in the malabsorption of nutrients required for health and growth. CD is difficult to diagnose because it resembles several other conditions that can cause malabsorption. The immune system of an individual with CD may be identifying gluten as a foreign substance and producing elevated levels of antibodies to rid the body of it. Most individuals with CD have celiac disease-associated antibodies and specific pairs of allelic variants in two HLA genes—HLA-DQA1 and HLA-DQB1.

There have been various case reports in the medical literature demonstrating a link between CD and Addison’s disease. O’Leary et al. have shown, in a series of patients with Addison’s disease, that there is a higher co-morbidity with CD than in any previously studied endocrine condition. It is strongly suggested that individuals with Addison’s disease be tested for CD.

The diagnosis of CD is achieved by two specific and sequential criteria. First, prior to treatment, there needs to be shown...
typical biopsy changes in the proximal small intestine. Second, there needs to be a definite clinical and/or pathological response to certain foods in the diet. Foods that may contain gluten include breads, biscuits, cakes, pastries, pizza, pasta, sausage meats, and certain soups, gravies, breakfast cereals and sauces. Gluten-free foods include unprocessed fruit, vegetables, milk, eggs, rice, fish and meat.

In the future, new forms of treatment for CD may include the use of substrates that regulate intestinal permeability to prevent gluten entry across the epithelium, different forms of immunotherapy, the development of gluten-free grains by genetic modification and use of gluten-degrading enzymes.

**CD in Individuals of Irish Descent**

Population-based screening studies have demonstrated a higher prevalence of CD in Ireland than elsewhere in the world. This prevalence rate is slightly less than 1% of the population. Mylotte et al. demonstrated a high prevalence of CD in workers in Galway in 1973.

The genetic stock of the Irish people is thought to have been established approximately 5,000 years ago, before the arrival of the Celts. It is thought that the ethnic and genetic mix is homogenous and distinct relative to much of mainland Europe. From prehistoric times to recent history—the great potato famine of the 19th century—oats were the only cereal and source of gluten consumed by most of the population of Ireland. Today, the Irish diet differs little from the rest of western civilization. Cronin states that until only very recently, the Irish diet has been almost gluten-free. He postulates that there is a suggestion that the possession of CD genes in areas of low-gluten consumption must confer a survival advantage and thus explains the high prevalence of CD in the modern-day Irish, who have only recently been exposed to such a wide variety of food with a high gluten content.

**Dental Significance of CD**

Numerous authors have stated there is an increased prevalence of dental issues in patients with CD. These oral manifestations are dental enamel defects and recurrent oral aphthae. Macroscopic mucosal changes have been found in as many as 40% of patients with CD. It has been postulated that these dental issues may provide a clue that could aid in the diagnosis of CD, which has historically proven hard to identify.

The enamel defects seem to be linked to the time course of odontogenesis. Aine stated that in children with CD, the central
incisors are always affected. Aine also states that symmetrically and chronologically distributed enamel defects are strongly associated with CD. Other studies have demonstrated the elevated prevalence of enamel defects in both incisors and molars. The defects are usually bilateral and symmetrical in the affected stages. These affected teeth share parallel developmental stages. The cause of these dental enamel defects is unknown. Patients with CD do not show a greater susceptibility to caries. Table 2 shows the dental and oral manifestations of CD.

It is apparent that dentists have a unique ability to aid in the diagnosis of an ailment that has puzzled diagnosticians for decades. The existence of a pattern of clear, symmetrical, chronological enamel defects has been well documented. Dentists who suspect CD from the dental and oral manifestations should refer the patient to the proper medical professional for a definitive diagnosis.

**Pathophysiology**

A unique feature of CD, compared to other autoimmune diseases, is the knowledge of a definitive environmental prompt (gluten) that is required for the disease to occur. Gluten and other proline-rich proteins are poorly digested in the normal human small intestinal tract due to a lack of prolyl endopeptidases. This can lead to gluten peptides as much as 10 to 15 amino acids in length. Gliadin is a glycoprotein extract from gluten that is believed to affect the enterocytes of individuals afflicted with CD, mainly via the overexpression of IL-15 in the intestine.

Another key component of the disease is the tissue enzyme transglutaminase. It is believed that if transglutaminase is not activated, gliadin is less immunogenic and may not stimulate T cells as effectively. When activated, this enzyme is important in the pathogenesis of CD in that the enzyme crosslinks ingested gliadin and causes specific deamidation of glutamine into glutamic acid and gliadin peptides. When such deamidation occurs, the gliadin peptides can be presented in a more effective manner to gliadin reactive CD 4 T cells, thereby increasing its immunogenicity.

Histologically it has been shown that two and a half hours after the ingestion of wheat gluten, there is a deterioration of villus architecture, distortion of microvillus structure, disorganization of the intermicrovillus pit region, and an increase in lysosome-like bodies in the apical cytoplasm of the luminal enterocytes, as well as pronounced hypertrophy of the rough endoplasmic reticulum of these cells. Also, at high magnification, CD patients exhibit enterocytes that are irregular in size and shape, with a decrease and disruption of the glycocalyx. Reductions in the length and density of the microvillus were also clearly identified.

The Marsh classification system is used to organize histology findings and is grouped into three categories: infiltrative, hyperplastic or destructive. These three categories share the common features of increased intraepithelial lymphocytes, decreased enterocyte height, crypt hyperplasia and villous atrophy to different degrees.

---

**TABLE 1**

Systemic Manifestations of Celiac Disease

<table>
<thead>
<tr>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood changes</td>
</tr>
<tr>
<td>Bloating</td>
</tr>
<tr>
<td>Joint pain</td>
</tr>
<tr>
<td>Skin rash</td>
</tr>
<tr>
<td>Reduced fat padding in feet</td>
</tr>
<tr>
<td>Amenorrhea</td>
</tr>
<tr>
<td>Iron-deficiency anemia</td>
</tr>
<tr>
<td>Dehydration</td>
</tr>
<tr>
<td>Weight loss</td>
</tr>
<tr>
<td>Muscle cramping in hands and legs</td>
</tr>
<tr>
<td>Difficulty gaining weight in children</td>
</tr>
<tr>
<td>Acidosis</td>
</tr>
<tr>
<td>Back pain</td>
</tr>
<tr>
<td>Dry skin</td>
</tr>
<tr>
<td>Night blindness</td>
</tr>
<tr>
<td>Bone disease</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
</tr>
<tr>
<td>Weakness</td>
</tr>
<tr>
<td>Edema</td>
</tr>
<tr>
<td>Electrolyte depletion</td>
</tr>
<tr>
<td>Diarrhea</td>
</tr>
<tr>
<td>Weakness</td>
</tr>
<tr>
<td>Abdominal discomfort</td>
</tr>
<tr>
<td>Abdominal distention</td>
</tr>
<tr>
<td>Lactose intolerance</td>
</tr>
<tr>
<td>Other autoimmune disorders</td>
</tr>
<tr>
<td>Recurrent fetal loss</td>
</tr>
<tr>
<td>Short stature</td>
</tr>
<tr>
<td>Delayed puberty</td>
</tr>
<tr>
<td>Osteoporosis/osteopenia</td>
</tr>
<tr>
<td>Epilepsy</td>
</tr>
<tr>
<td>Attention-deficit disorder</td>
</tr>
<tr>
<td>Migraines</td>
</tr>
<tr>
<td>Acidosis</td>
</tr>
<tr>
<td>Back pain</td>
</tr>
<tr>
<td>Dry skin</td>
</tr>
<tr>
<td>Night blindness</td>
</tr>
<tr>
<td>Bone disease</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
</tr>
<tr>
<td>Weakness</td>
</tr>
<tr>
<td>Edema</td>
</tr>
<tr>
<td>Electrolyte depletion</td>
</tr>
<tr>
<td>Diarrhea</td>
</tr>
<tr>
<td>Weakness</td>
</tr>
<tr>
<td>Abdominal discomfort</td>
</tr>
<tr>
<td>Abdominal distention</td>
</tr>
<tr>
<td>Lactose intolerance</td>
</tr>
<tr>
<td>Other autoimmune disorders</td>
</tr>
<tr>
<td>Recurrent fetal loss</td>
</tr>
<tr>
<td>Short stature</td>
</tr>
<tr>
<td>Delayed puberty</td>
</tr>
<tr>
<td>Osteoporosis/osteopenia</td>
</tr>
<tr>
<td>Epilepsy</td>
</tr>
<tr>
<td>Attention-deficit disorder</td>
</tr>
<tr>
<td>Migraines</td>
</tr>
</tbody>
</table>

**TABLE 2**

Oral and Dental Manifestations of Celiac Disease

<table>
<thead>
<tr>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fissured tongue</td>
</tr>
<tr>
<td>Cheilitis</td>
</tr>
<tr>
<td>Aphthous stomatitis</td>
</tr>
<tr>
<td>Delayed tooth eruption</td>
</tr>
<tr>
<td>Diminished size of teeth</td>
</tr>
<tr>
<td>Salivary gland dysfunction</td>
</tr>
<tr>
<td>Problems in enamel formation</td>
</tr>
</tbody>
</table>

---
Immunohistochemistry of the small intestine of patients shows villous atrophy, crypt hyperplasia and elevated levels of intraepithelial lymphocytes. These intraepithelial lymphocytes are localized between intestinal epithelial cells at the basolateral side of the epithelium and are thought to play an important role in immunosurveillance of the epithelium. In the lamina propria, CD4 cells elicit an immune response and intraepithelial lymphocytes acquire activating natural killer receptors and the ability to lysis stressed epithelial cells independent of T-cell receptor signaling, which likely contributes to the typical tissue damage seen in celiac disease.

Conclusion

CD is often misdiagnosed and can cause the patient much suffering. Once diagnosed, the individual can live a happy and full life. As in the case of many other systemic disorders, the dentist plays a unique and vital role in diagnosing CD in their patients. In this regard, the dentist can provide a service to the patient, which can tremendously increase the patient’s quality of life.

Queries about this article can be sent to Dr. Maloney at wjm10@nyu.edu.

References

A B S T R A C T

The aim of this report is to describe the positive effect of plasma-rich in growth factor (PRGF) on pulp regeneration and apex formation in cases with necrotic pulps and open apices. After access cavity preparation and cleaning of the canal, triple antibiotic paste was inserted into the canals for the purpose of disinfection. After two weeks, apical bleeding was mechanically created by insertion of a #80 file through the apex. PRGF obtained from the patient was centrifuged and injected into the canals up to the level of the cementoenamel junction; the teeth were restored temporarily. The patients returned for review two weeks later. If there was absence of pain, swelling, fistula or any other complication, the teeth were sealed with MTA and composite. At 22 months follow-up, complete apex closure in two teeth and apical closure and continued increase of dentinal wall thickness in two other cases were evident.

Endodontic treatment of teeth with necrotic pulps and open apices can be a great challenge for practitioners, because it is very difficult to achieve complete cleaning/filling and a tight seal with traditional techniques and materials. Moreover, these teeth have thin dentinal walls, which leaves them susceptible to subsequent fracture after endodontic treatment.1-3

As stated by Torabinejad and Turman,4 “the ideal outcome for a tooth with an immature root and necrotic pulp would be the regeneration of pulp tissue into a canal capable of promoting the continuation of normal root development.” Thus, regenerative endodontics may be a suitable alternative for conventional root canal treatment in teeth with necrotic pulps and open apices.4-6

Revascularization of immature avulsed teeth has been well-documented. However, it has also been shown that infection may arrest the progress of revascularization and continued development of the root.7,8 And it has been indicated that even in the presence of a large periapical lesion, vital pulp tissue and Her-twig’s epithelial root sheath are possible.9,10 This tissue could proliferate if canal cleaning and disinfection is performed thoroughly and the previously existent inflammatory process is reversed.8,11

The thickening of dentinal walls and apex maturation may be observed in teeth with necrotic pulps and open apices.1,4,8,10,12,13

To increase the success rate of a pulp regeneration procedure, use of a suitable physical scaffold for supporting cell growth and

The Plasma-Rich in Growth Factor as a Suitable Matrix in Regenerative Endodontics

A Case Series

differentiation is mandatory. For example, platelet-rich plasma (PRP) has been used for this procedure and seems to be a suitable material.\textsuperscript{2,4,14}

Another scaffold that might be ideal for this procedure is plasma-rich in growth factor (PRGF). It has been shown to be enriched with PDGF, TGF\_1, TGF\_2, FGF, VEGF, EGF and ILGF\_1.\textsuperscript{12,15} Considering the effect of PRGF on Hertwig’s epithelial root sheath and the increase of alkaline phosphatase activity, acceleration of osteogenic differentiation of human dentinal stem cells seems to be possible.\textsuperscript{16} It has been hypothesized that after root canal disinfection with a paste mixture of three antibiotics, formation of the blood clot would serve as a matrix that could speed up the differentiation of the remaining progenitor cells.\textsuperscript{17}

This case series describes the effect of PRGF on pulp regeneration and apex formation in four cases with necrotic pulps and open apices in children 6 to 11 years old. All treatment procedures were performed in the Endodontic Department, Faculty of Dentistry, Islamic Azad University, Tehran, Iran. The medical history of all patients was non-contributory.

**Case One**
The patient was an 11-year-old girl with a history of trauma to both maxillary central incisors. During extraoral examination, an acute apical abscess with swelling at the anterior area of the maxilla was present. On intraoral examination, no soft tissue abnormality was present. Both incisors were sensitive to percussion and palpation tests. Cold test using refrigerant spray (Coltène/Whaledent, Switzerland) and the electric pulp test (Parkell, New York) induced no response on either incisor. Control teeth had no sensitivity to percussion and palpation; however, the response to the cold test was positive. During periodontal examination, no periodontal pocket was observed. A periapical radiograph showed both central incisors had immature roots, thin dentinal walls and open apices (Figure 1A).

On the basis of these findings, the pulpal diagnosis was necrosis and the periapical diagnosis was acute apical abscess for both teeth. Abscess drainage was done via labial mucosa without antibiotic prescription. The common treatment protocol used for all patients presented in this case series (application of PRGF for a regenerative endodontic procedure) was applied to both incisors.

**Case Two**
The patient was an 8-year-old girl with a history of trauma (three months earlier) to the right central incisor. On extra- and intraoral examination, everything was normal. Both incisors were sensitive to percussion and palpation tests. Cold test and electric pulp test induced no response on the right central incisor. Control teeth had no sensitivity to percussion and palpation; in addition to a positive response to cold test. During periodontal examination, no periodontal pocketing was observed. A periapical
radiograph showed that both central incisors had thin dentinal walls and open apices (Figure 2A).

On the basis of these findings, the pulpal diagnosis was necrosis, and the periapical diagnosis was acute apical periodontitis. The common treatment protocol was performed for this patient.

Case Three
The patient was an 8-year-old girl with a history of trauma to the anterior area of the maxilla. The patient’s parents reported that her right central incisor had been avulsed in an accident four months earlier. No replantation had been done for this tooth. On extra- and intraoral examination, everything was normal. The left central incisor was sensitive to percussion. Cold test and electric pulp test induced no response on this incisor. Control teeth had no sensitivity on percussion and palpation, in addition to the positive responses to the cold test. No periodontal pocketing was present. A periapical radiograph showed the left central incisor had thin dentinal walls and an open apex (Figure 3A).

On the basis of these findings, pulpal diagnosis was necrosis, and the periapical diagnosis was acute apical periodontitis. The common treatment protocol was performed for this patient.

Common Treatment Protocol
Prior to treatment, the patients’ parents were told that the treatment protocol was an attempt to help apex maturation and thickening of the dentinal walls. They were also told that the success of this treatment is not definite. They signed the informed consent form.

The patients received local anesthesia of 2% lidocaine with 1:80,000 epinephrine (Darou Pakhsh, Tehran, Iran), and the teeth were isolated with a rubber dam. An access cavity was prepared using fissure diamond burs (Dentsply/Maillefer, Tulsa, OK). A new bur was used for each tooth. In all patients, no hemorrhage was observed on entering the root canals. The working length of each canal was determined using a #80 K-file (Maillefer, Dentsply, Switzerland) and a periapical radiograph. The canals were irrigated with 8 ml of 0.5% sodium hypochlorite (NaOCl), followed by normal saline prior to placement of triple antibiotic paste (consisting of 500 mg metronidazole, 100 mg minocycline and 200 mg ciprofloxacin) to avoid the unwanted interaction of antibiotics with NaOCl.

After drying the canals with sterile paper points (Ariadent Co, Tehran, Iran), a thick paste mixture of triple antibiotics was prepared. For each case, a freshly prepared paste was used. This paste
was prepared from the powder after removal of the sugar coating with a scalpel blade. The pills were crushed individually in separate mortars and each antibiotic ground to a fine powder. Equal amounts of antibiotics (1:1:1) were combined on a mixing pad. The powders were then mixed with an MP carrier (equal amounts of macrogel ointment and propylene glycol) with the proportion of 1:6 (1 part MP to 6 parts mixed antibiotics). The antibiotic mixture was inserted in the coronal two-thirds of the canal using a carrier (Q-106, Beijing, China) and packed using #80 paper points (Ariadent Co, Tehran, Iran). For the temporary sealing of the access cavity, Cavit (ESPE, Seefeld, Germany) was used.

For preparation of PRGF, 10 ml of patient blood was poured into sterile tubes containing 5 ml of 3.8% sodium citrate. The blood-filled laboratory tubes were centrifuged with 460 g speed for 8 minutes. Plasma was consequently divided into different layers. The third layer formed in the tube precisely on top of the red blood cells layer. It contained x4 time concentration PRGF, of which 0.5 ml was used for this study. 0.5 ml of CaCl2 was added to the separated solution to activate release of the growth factors.

After two weeks, all patients were asymptomatic. At this appointment, triple antibiotic paste was removed and apical bleeding was mechanically created by inserting a #80 file to within 1 mm of the radiographic apex. The PRGF obtained from the previously centrifuged patient’s blood was injected into the canals to the level of the cemento-enamel junction. This was done to help position the growth factors to the apical portion of the canals. The teeth were temporarily restored using Cavit (ESPE, Seefeld, Germany).

The patients returned for follow-up two weeks later. In the absence of pain, swelling, fistula or any other complication, the teeth were sealed with MTA (ProRoot, Dentsply, USA) and composite (3M ESPE, Ontario, Canada), a thin layer of MTA at the floor of the cavity, and composite for restoring the cavity.

Follow-up sessions at 3, 6, 9 and 22 months were scheduled for all patients so the apical closure could be evaluated. At the first three-month follow-up, increased dentinal wall thickness in all four cases and apical closure in at least one case were visible (Figures 1B, 2B, 3B). At the six-month follow-up, an increase in dentinal wall thickness was evident in one case and apical closure in all other cases (Figures 1C, 2C, 3C). At the nine-month radiographic follow-up, resolution of the periapical lesion, apical closure and continued increase of dentinal wall thickness in both incisors of Case 1 were evident (Figure 1D).

An increase in dentinal wall thickness was evident in Case 2 (Figure 2D). And increasing thickness in the dentinal walls and apical closure were observed in Case 3 (Figure 3D). All three patients remained asymptomatic.

At the 22-month follow-up, complete closure of the apex in Case 1 (Figure 1E), apical closure and continued increase of dentinal wall thickness in Case 2 (Figure 2E) and Case 3 (Figure 3E) were evident.

**Discussion**

Mechanical instrumentation of canals with necrotic pulps and open apices is futile in immature teeth because of the weak dentinal walls, so disinfection of these canals is restricted to irrigation and intracanal medicament. This may induce regeneration of pulp tissues in these teeth and thereby increase the likelihood of the root length and apical closure. The cases presented here can be added to previously reported cases to form the basis for future studies.

For successful regenerative endodontics in teeth with necrotic pulps and open apices, the removal of infection from the canal, a hermetic coronal seal, a physical scaffold for the promotion of growth/differentiation and some molecules for stimulation of stem cells are mandatory. In the cases presented here, disinfection was obtained with NaOCl and triple antibiotic paste, as proposed by other researchers.

Banchs and Trope studied revascularization of immature necrotic but adequately disinfected premolars with a suitable matrix. The triple antibiotic paste was used. Apical closure, increased dentinal wall thickness, and the disappearance of both periradicular radiolucency and sinus tract were evident at follow-up. Chueh et al. studied 23 teeth with necrotic pulps and open apices where canals were irrigated with 5.25% NaOCl without mechanical instrumentation. Calcium hydroxide was used, and canals were filled only if they remained symptomless at follow-up. All teeth with necrotic pulps and open apices showed signs of root formation within 10 to 20 months (mean 16 months). Since 2.5% NaOCl affects vital pulp and periapical cells, 0.5% NaOCl was used in this study, but with an increased volume, as suggested by Banchs and Trope.

A component of tissue engineering with great importance is using a physical scaffold. This scaffold should be able to provide a correct position of cell location and also regulate differentiation. These scaffolds may be natural or synthetic. Although most studies have considered only the blood clot as the scaffold, PRP has been introduced as a suitable scaffold.

In the cases presented here, PRGF was used as the scaffold. PRGF is a combination of autologous proteins prepared from PRP. It provides platelet-enriched plasma that contains no leukocytes. It may be considered a natural growth factor composite to help endodontic regeneration. It has important advantages. It is an autologous product, and so avoids the risk of disease transmission. It can facilitate the simultaneous action of growth factors and provide a safe and effective biocompatible agent that can be resorbed by the body after initiating local regeneration. It may limit inflammation, suppress cytokine release and induce tissue regeneration. However, PRGF has only limited potential in local bone formation in animals.

Although the number of cases presented here is small, it’s possible that pulp regeneration in teeth with necrotic pulps and open apices is predictable if the suggested protocol is followed.
In these circumstances, PRGF may be a suitable scaffold for pulp regeneration. Additional clinical trials may further assess the suitability of PRGF for pulp regeneration. More research on the origin and cellular nature of the newly formed tissue would be beneficial.

Queries about this article can be sent to Dr. Jafarzadeh at hamid_j365@yahoo.com, or JafarzadehBH@mums.ac.ir

REFERENCES