Bimaxillary Full Arch Fixed Dental Implant Supported Treatment for a Patient With Renal Failure and Secondary Hyperparathyroidism and Osteodystrophy

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A long-term dialysis patient with end-stage renal disease (ESRD) also referred to as chronic kidney disease (CKD) due to IgA nephropathy complicated by severe secondary hyperparathyroidism and renal osteodystrophy was successfully treated with dental implant-supported fixed prostheses. Phosphate binders, vitamin D, calcium cinacalcet calcimimetic therapy, and dialysis 3 times weekly had been instituted with standard divalent ion serum assessments. Successful control of the patient's secondary hyperparathyroidism was achieved. Long and wide diameter implants were used with an anterior guidance occlusion scheme to reduce the per-square-millimeter off-axial implant force delivered to the bone. Patients with ESRD and renal osteodystrophy may be successfully surgically and prosthetically treated with long wide dental implants supporting fixed full arch splinted dental prostheses with an appropriate occlusal scheme.

Key Words: dental implant, renal disease, kidney dialysis, cinacalcet, calcimimetic therapy, osseointegration, osteodystrophy, chronic kidney disease

Introduction

Technological and medical advancements have allowed many patients with chronic illnesses to maintain an active and productive life that includes dental care. Patients with end-stage renal disease (ESRD) also referred to as chronic kidney disease (CKD) can be sustained with dialysis on an out-patient basis for many years. In 2007 there were 527 283 patients in the USA under treatment for end-stage renal disease receiving dialysis.[1] Many of these patients require kidney transplants, but there may be a prolonged wait for a suitable donor.[1] During this time these patients may seek dental care. Many of these patients may have their treatment needs met with dental implant supported prostheses. The case presented herein demonstrates that complete bimaxillary extraction and immediate implant placement may be successfully performed with a satisfactory outcome.[2] This expedited treatment approach may reduce surgical trauma and treatment time.

The objective of this article is to report the treatment of a patient with end-stage renal disease due to biopsy proven immunoglobulin A (IgA) nephropathy complicated by severe secondary hyperparathyroidism successfully medically treated and with implant supported fixed complete dentures.

Case Report

A 34-year-old male patient was referred to one of the authors (DF) by Donated Dental Services for pro bono treatment. The patient had been diagnosed with immunoglobulin A (IgA) nephropathy at age 19 by percutaneous renal biopsy. He had a long history of tobacco use. Over the next several years, during which he was intermittently compliant with medical follow-up, he developed hypertension and secondary hyperparathyroidism. Two years after being lost to medical follow-up, he presented to a local community hospital at age 27 with a creatinine of 28 mg/dL (average male: 0.7–1.2mg/dL). Hemodialysis was initiated three times weekly, per standard treatment regimens. Due to noncompliance with dietary restrictions and phosphate binder therapy, he developed severe secondary hyperparathyroidism and associated renal osteodystrophy. His peak intact parathyroid hormone (iPTH) assay level was approximately 3000 pg/dL (average: 10–60 pg/mL). Target levels are 150–300 ng/dL. His medical course was complicated by a bicycle accident during which he suffered bilateral compound ulnar and radial fractures that required open repair, and received 2 blood transfusions. Despite improved dietary and phosphate binder compliance, his iPTH levels remained markedly elevated. With the introduction of cinacalcet, a novel calcimimetic for the treatment of secondary hyperparathyroidism, his iPTH levels gradually fell to the target range where they have remained quite stable for the last 2 years. Though referral for renal transplantation had been made soon after starting dialysis, he was not eligible for a transplant because of his severely carious teeth and dental infections of the jaws (Figures 1 through 4). At the time of referral for definitive dental care he was taking Nephrocaps vitamin supplement, amlodepine besylate (Norvasc) 5 mg/qd, cinacalcet (Sensipar) 30 mg/qd, metoprolol (Lopressor) 50 mg/bid, and paroxetine (Paxil) 20 mg/qd. Hypertension had developed secondary to the nephropathy. He has had 2 blood transfusions, 3 and 7 years prior. He has a long history of tobacco use and currently smokes less than a

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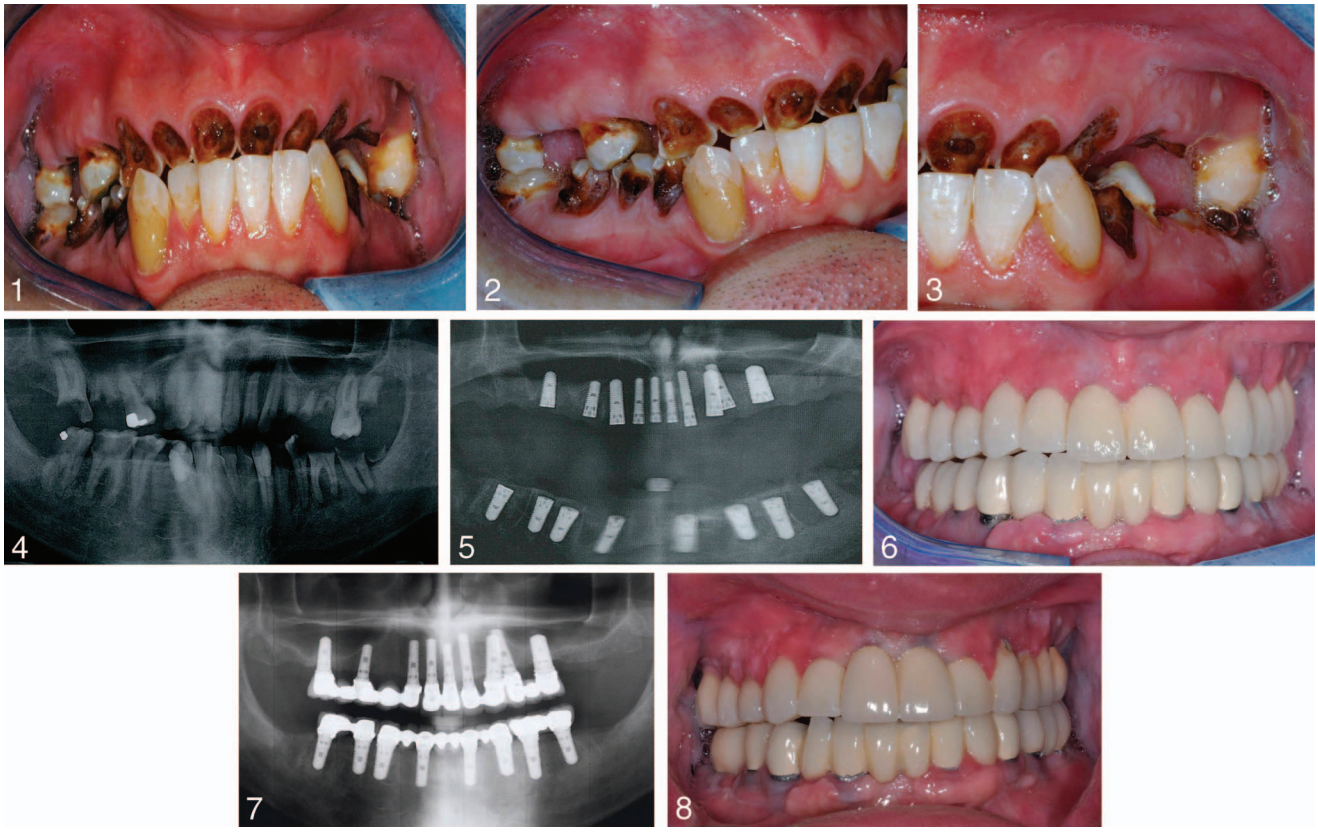

Figures 1–8. **Figure 1**. Preoperative frontal view. **Figure 2**. Preoperative right side. **Figure 3.** Preoperative left side. **Figure 4.** Preoperative panoramic radiograph. **Figure 5**. Postoperative radiograph of bimaxillary placed implants. **Figure 6**. Frontal view of cemented prostheses. **Figure 7**. Panoramic radiograph of cemented prostheses. **Figure 8.** Frontal view after 2 years of uneventful function.

pack of cigarettes each day. He was strongly advised to quit by his health care providers. He requires a 2000-mg amoxicillin premedication for dental treatment to reduce the risk of infection of his indwelling shunt. The patient presented for treatment and was examined clinically and radiographically (Figures 1 through 4). Full-mouth radiographic series, a panoramic and a bimaxillary cone beam computerized scan and mounted study casts were made. The patient's internist/nephrologist (MM) was consulted by telephone. The patient's carious teeth had delayed his candidacy for a kidney transplant. It was strongly suspected that the fall during which the patient sustained bilateral forearm bone fractures was indicative of poor bone mineralization, particularly given the previously uncontrolled secondary hyperparathyroidism. The carious roots of teeth #13 and 14 were extracted and fragments of inter-radicular bone were sent to the University of Connecticut, Department of Oral Pathology for examination and found to have normal osseous microscopic anatomy. The osseous texture of the bone during the extraction at this site was sensed to be very dense and unyielding to the surgeon (DF). This was deemed to indicate a somewhat supportive quality for implant treatment. After the appropriate discussions, a judgment was made to proceed with implant treatment.

This patient has a history of noncompliance and this may affect his longevity and the long-term outcome of the implant treatment. The patient was counseled on this issue and agreed to comply with our instructions.

A literature search for case management background was made on the National Library of Medicine's website PubMed and articles were retrieved using the keywords IgA nephropathy, parathyroid, hyperparathyroidism, and dental implant. A compilation of published data was made but there has been no published implant treatment of patients with end-stage renal disease. Treatment options were discussed with the patient and his physicians and a definitive plan was made for fixed bimaxillary porcelain fused to metal implant-supported complete dentures. Most of the maxillary teeth were carious to or below the osseous margins and the three salvageable maxillary teeth would not be appropriate for the chosen treatment plan. All of the maxillary teeth were planned for extraction. An implant-supported fixed porcelain fused to metal maxillary complete denture was finally chosen. In the maxilla, 1 implant per tooth was decided to minimize the per-square-millimeter of occlusal force applied to the bone via the prostheses and the implants. All of the posterior mandibular teeth were also carious to or below the osseous margins and deemed unrestorable and planned for extraction. The mandibular anterior teeth #22 through 27 were unscathed by caries or periodontitis but were somewhat rotated and crowded. These teeth were to be left untreated since the patient declined orthodontic treatment for these teeth, but since caries was the patient's primary problem, the decision was made to remove these teeth to prevent future caries, overload complications, and to simplify the occlusal scheme. An implant-supported

fixed porcelain fused to the metal complete denture was decided for the final mandibular prosthesis. A goal of 1 implant per tooth was decided upon to maximize the bone to implant interface surface, but osseous conditions and implant proximity precluded this plan in some sites. An interim complete maxillary immediate denture was constructed. On the day of surgery the patient was premedicated with 2000 mg amoxicillin per os, his vital signs recorded and monitored, and administered infiltration local anesthesia (articaine 4% with 1:100 000 epinephrine Septocaine). A pulse oximeter monitored the patient's blood oxygen saturation for all surgical appointments and was at 97–98 % at all times. All the maxillary dentition was atraumatically extracted to preserve the osseous architecture for the dental implants (Implant Direct, Ventura, Calif) that were immediately placed in sites #3, 4, 6, 8, 9, 11, 13, 14. The implant sites were first drilled with a pilot drill and then successive round osteotomes were used to expand the osteotomy and compress the bone to increase density for initial implant stability. All of the implants were placed 1–2 mm below and approximately 2 mm lingual to the facial osseous rim. At site #6 a larger 4.7-mm diameter implant was placed due to inadequate stability of a 3.7-mm diameter implant thus not attaining the desired 1.8-mm minimum facial clearance. The 4.7 $\times$ 13 implant placed at site #6, maxillary right cuspid, was less than 2.0 mm from the facial cortex but was left, in hopes of being used. The implant at site #13 was angled to the palatal to avoid a facial dehiscence. The deficient posterior maxilla was treated by cutting a cylinder of bone with a trephine and infracturing the cylinder so as to elevate the sinus floor. Dry medical grade calcium sulfate (DentoGen, Springfield, NJ) was introduced into the osteotomy by a 1-cc syringe that delivered the calcium sulfate to press the sinus lining gently and evenly superiorly. Approximately 1 cc of calcium sulfate was used. The calcium sulfate was delivered dry but became thoroughly soaked in blood during the delivery process. Medical grade calcium sulfate (DentoGen) was placed in all the sites and the sockets covered with collagen plugs (Salvin) lay sideways to provide a thick covering and protection for all of the surgical wounds and to contain the calcium sulfate. 3–0 chromic gut suture (Ethicon, Johnson and Johnson, New Brunswick, NJ) was placed to hold the collagen plugs in place and contain the calcium sulfate. The immediate maxillary denture was adjusted and delivered. The patient was dismissed in good condition with no bleeding. The patient was seen the next day and was found to be doing well.

Four weeks later the mandible was premedicated and bilaterally locally anesthetized with 3.8-cc articaine (Septocaine) on each side and teeth #17–31 were atraumatically surgically extracted and dental implants (Implant Direct) were immediately placed in sites #19, 20, 21, 23, 26, 28, 29, 30. All voids in the sites were filled and augmented with calcium sulfate and covered with collagen plugs and sutured with 3–0 chromic gut. In both jaws, each implant was placed so that the implant platform was located approximately 1.0 mm below the osseous facial socket rim and approximately 2.0 mm lingual to the facial aspect of the facial cortex. There were no detected osseous dehiscences or fenestrations in the mandible. A mandibular provisional complete immediate denture was adjusted and delivered. The patient was assessed the following day and again in 1 week for suture removal.

Healing was uneventful after each surgery (Figure 5). The provisional complete dentures were adjusted and the intaglios soft lined as healing and tissue shrinkage progressed. The patient returned at 8 weeks for an assessment appointment to monitor healing. He was found to be healing well. There was no implant mobility or infection observed.

After 7 months of healing, the patient returned for the prosthetic phase. The patient was premedicated and the maxillary sites locally anesthetized with 5.4 cc articaine infiltration. The gingival overgrowth was removed with a medium coarse diamond. The #6 implant was found to have a 4-thread dehiscence and was removed to preclude any periimplantitis issues that may complicate the impending kidney transplant. The remaining abutments were placed and prepared for parallelism. The abutment screws torqued into place and the access holes filled with a compomer (Dyract, Dentsply, York, Pa) and light cured. A standard crown and bridge impression (Imprint, ESPE, St Paul, Minn). A light-cured bis-acryl (Triad) provisional fixed complete denture was made to approximate the proposed definitive restoration.

The mandible was treated 4 weeks later. A full and partial thickness apically positioned flap was raised exposing all the implants. The cover screws were removed and stock abutments were placed and slightly prepared extraorally for parallelism. The abutments were torque into position and the access holes filled with a light-cured compomer (Dyract). A polyvinyl siloxane impression was taken. Bleeding was controlled with local infiltration of articaine. Later, a saturated (16%) aqueous tranexamic solution oral rinse was administered to insure postoperative bleeding control. The vertical dimension was established with the removable complete dentures used as a guide. The centric relation was taken with a fast set polyvinyl siloxane (Futar). A full-arch bis-acryl splint was fabricated for a provisional restoration and occluded with the maxillary provisional in a group function to reduce the potential for material fracture that may occur in an anterior-guided scheme. A directly applied bis-acryl stent was applied to the facial gingival flap for protection and fixation. No sutures were placed. The ramp setting was set arbitrarily at 20° on the nonarcon semi-adjustable articulator (Hanau). Prosthetic splint grouping was established in the definitive restoration.

The stent was removed 1 week later and normal healing was observed.

At this point the patient was wait-listed for renal transplant surgery.

A segmented splinted porcelain fused to noble alloy complete fixed denture was ordered (York Dental Lab, Branford, Conn). The laboratory technician was instructed to apply extra die separator to the working dies to insure a passive fit. The prostheses replaced first molar to first molar occlusion in both arches. Conventional waxing, casting, frame try-in, remount, and porcelain stacking application techniques were used. The porcelain was applied to the frame and the prostheses were tried in and the anterior guidance occlusal scheme refined. The definitive dentures were provisionally cemented with zinc oxide eugenol (Opotow, Ft Collins, Colo) to assess function and esthetics for 2 weeks. The dentures were then definitively

cemented with a resin modified glass ionomer (FujiCEM, CG America, Alsip, Ill; Figures 6 and 7). The patient has been functioning uneventfully for 2 years (Figure 8).

Discussion

Generally, a glomerular filtration rate of less than 60 ml/min for 3 months or longer is referred to as chronic kidney disease. A glomerular filtration rate less than 15 mL/min/1.73 square meters is defined as stage V kidney disease and indicates the need for renal replacement therapy is imminent.[1] Diabetes is the most common comorbid condition of chronic kidney disease and causes about 40% of all chronic renal failure (Brockman).

IgA nephropathy is an immune complex disease of the glomerulus of the kidney that occurs more frequently in males than females and occurs in all age groups.[3] These patients most commonly have hematuria and proteinuria and have no symptoms, and this can result in a delay of treatment. IgA nephropathy can only be diagnosed through immunofluorescence staining of a renal biopsy specimen. This diagnosis is required for the proper management of patients with glomerular disease, particularly when there may be progression to end-stage renal failure. This occurs in as much as 20% of patients with IgA nephropathy. An accurate diagnosis is important since the disease can recur after a transplanted kidney, but this does not usually cause the transplant to fail.[4]

As renal function deteriorates, phosphorous excretion falls, causing a rise in serum phosphate levels. Simultaneously, serum calcium concentration declines, activating a cascade of events that stimulates parathyroid hormone release that maintains normal phosphorous excretion through increased renal phosphate excretory mechanisms, and maintains normal serum calcium levels via calcium mobilization from the vast bony reservoir. Unfortunately, this is a maladaptive mechanism that results in a condition known as renal osteodystrophy (also known as metabolic bone disease). These events occur in the majority of patients on dialysis. There are several forms of renal osteodystrophy, including increased bone turnover typical of hyperparathyroidism, osteomalacia as seen in aluminum toxicity, mixed bone disease, and adynamic (low turnover) bone disease. Increased bone turnover is associated with increased osteoid and but reduced mineralization. When the disease becomes severe, bone is resorbed and replaced with fibrous tissue and woven bone, and is termed "osteitis fibrosa cystica." It may even take on an appearance of a tumor on plain radiographs, known as a Brown's tumor. While a double tetracycline labeling bone biopsy provides the most reliable method of classifying the type of bone disease, the majority have high turnover, PTH-dependent disease, or adynamic (low turnover) disease. In clinical practice, diagnosis and treatment rely on monthly measurement of calcium and phosphorus, and quarterly assays of intact PTH and 25-hydroxy-vitamin D levels. These data are used to guide prescribing of oral phosphate binders consumed with meals, low phosphate diet, vitamin D analogues, and cinacalcet therapy when deemed appropriate.[5]

Secondary osteopathies can occur. A bone biopsy is usually done to classify patients into hyperparathyroid, osteomalacia, mixed, and adynamic types.[6] Metabolic bone diseases generally result from a disorder of bone remodeling. Mild hyperparathyroidism results in an increase in bone turnover. This turnover can be measured by double tetracycline labeling test expressed as per unit of bone surface. The increased bone turnover is usually accompanied by hypomineralization. In prolonged hyperparathyroidism there is bone loss demonstrated by thinning of the cortical bone from the endosseous aspect. In patients with severe hyperparathyroidism bone is resorbed and replaced with fibrous tissue and woven bone, osteitis fibrosa. In patients with osteitis fibrosa, the larger the bone surface, the less mineralization of any newly formed bone. Osseous formation is lower in patients with osteitis fibrosa.[6]

Parathyroid gland

The parathyroid glands are located in the paraposterior positions of the thyroid gland.[7] There are generally 4 glands, 2 on each side, measuring 6-mm long, 3–4 mm across, and 1–2-mm thick. They were first identified by Viktor Sandstrom in 1880. The glands derive their blood supply from the inferior thyroid artery. There are 2 populations of cells, chief cells, and oxyphil cells. The chief cells produce parathyroid hormone, parathormone. The function of the oxyphil cells remains unclear. Parathormone, also known as intact PTH, is an 84 amino acid polypeptide that is derived from the cleavage of the 115 amino acid polypeptide, Pre-Pro-PTH. The primary function of parathormone is to maintain serum calcium and phosphorous levels within the narrow range necessary for proper cellular function, particularly muscle and nerve cells, but it also plays a significant role in the coagulation cascade, and cellular hormonal release. It is therefore of utmost importance to maintain calcium concentrations within the proper range. The chief cells of the parathyroid gland have exquisitely sensitive surface receptors, known as calcium-sensing receptor (CaSR). In response to falling calcium levels, parathormone directly stimulates renal tubular calcium resorption and bone calcium release. In sustained hypocalcemia, parathormone promotes precursor 25-hydroxy-vitamin D conversion to active 1,25 dihydroxyvitamin D, known as calcitriol, by renal tubular cells. Hypercalcemia has the converse effect. 25-Hydroxyvitamin D is derived from dietary sources and skin production upon exposure to ultraviolet wavelengths. In addition, calcitriol promotes gastrointestinal calcium absorption, bone calcium release through osteoclast activation, and renal tubular reabsorption of filtered calcium, to maintain serum calcium concentrations within the ideal range. In addition, parathormone promotes renal phosphate excretion. Bone matrix is generally composed of collagen fibers with calcium phosphate (85%), calcium carbonate (10%), and calcium and magnesium fluoride (5%).[7]

Hyperparathyroidism

In primary hyperparathyroidism, typically 1 of the 4 glands transforms into an adenoma that functions autonomously and releases parathormone independent of serum calcium levels. In its early asymptomatic stage, this condition is commonly detected upon routine blood testing that reveals an elevated calcium level (10–12 mg/dL, normal: 9–10.5 mg/dL). However, as calcium rises beyond 12 mg/dL, symptoms include

depression, fatigue, polyuria, polydipsia, dehydration, anorexia, nausea, constipation, and muscular weakness, followed by neuropsychiatric symptoms, confusion, and even death. Levels of parathormone can be measured with a radioimmunoassay that measures the intact PTH molecule. When hypercalcemia occurs in the presence of elevated intact PTH levels, this is termed primary hyperparathyroidism. The hyperfunctioning gland can be detected with a nuclear medicine technetium sestambi scan. Once identified, the adenomatous gland can be surgically removed.[8,9]

Primary hyperparathyroidism

Primary hyperparathyroidism is a systemic hypercalcemic disease that results in persistent bone resorption and can result in reduced radiographic dental lamina dura, decreased osseous cortical density, and an increased prevalence of oral osseous tori.[8,9] Padbury and colleagues studied the effects of primary hyperparathyroidism on the oral cavity.[8] They found that there may be a widening of the periodontal ligament (PDL), which may correlate to the patient's parathyroid hormone serum level. Hyperparathyroidism is not associated with an increased risk for periodontitis and does not cause attachment loss. However, it is associated with an increased occurrence of tori and a reduction of lamina dura, interdental alveolar bone density, and cortical density. There is also can be an associated cortical thinning at the angle of the mandible. In this disease there is a loss of skeletal cortical bone and a preference for trabecular bone formation. Patients with a widened PDL width generally also had tori irrespective of age or gender. They did not find a severe loss of lamina dura, brown tumors, or an osseous ground glass radiographic appearance.

Generally, only 1 of the 4 glands is in hyperfunction, and it can be identified with a nuclear medicine technetium sestamibi scan. The hyperfunctioning gland can then be surgically removed allowing the remaining glands to adequately take over normal hormone production.

Primary hyperparathyroidism is diagnosed by immunoassay of the hormone. The hormone level is high even with a high calcium serum level. Additionally, phosphate serum, bone density and vitamin D levels can be assessed.[10] In hypoparathyroidism the patient may feel fatigue and anxiety, while hyperparathyroidism may have no symptoms.

Secondary hyperparathyroidism

With chronic kidney disease there is phosphate retention, decreased renal synthesis of active vitamin D (calcitrol) with resultant hypocalcemia and secondary hyperparathyroidism. If untreated, osteodystrophy (osteitis fibrosa cystica) develops.[1] Early control of serum phosphate is essential to slow the progression of the disease but decrease the risk for development cardiovascular disease.[1]

Secondary hyperparathyroidism can occur with vitamin D deficiency, long-term lithium therapy, and chronic renal disease. Osteoclastic remodeling then occurs, causing renal osteodystrophy. Chronic renal disease causes a low calcium serum level that induces that induces parathormone production. Secondary hyperparathyroidism is diagnosed also by immunoassay, but the hormone level is high and the calcium serum level can be low or normal. Alkaline phosphatase levels are usually increased in secondary hyperparathyroidism due to chronic renal disease.

IgA nephropathy is an immune-complex glomerular renal disease and occurs at all ages with a higher occurrence in males. These patients generally present with asymptomatic hematuria and proteinuria.[3,9] Prednisone and antihypertensive treatments significantly improve proteinuria and attenuate renal function deterioration.[11]

Leontiasis ossea is a condition of hypertrophy of the facial bones that can result from secondary hyperparathyroidism from chronic renal failure.[12] The patient treated herein did not have any signs of facial hypertrophy, which nay have dramatically affected the implant treatment.

Rationale for dental implant treatment

Skull bones such as the maxilla and mandible form in a different embryologic mechanism than the long bones.[7] Maxillary and mandibular bones may not be affected by the same pathophysiology as the long bones. The patient here fell from his bicycle and fractured his right and left ulnae and radii. The weakened cortical bone fractured from the sudden traumatic force from the fall. However, the loads delivered to the bones of the jaws by dental implants are not usually sudden and of such high magnitude. Occlusal loads are frequent, cyclical and multidirectional but the designed occlusal scheme constructed generally directs the forces axially to the implants. This may produce an osseous microstrain that induces apposition.[13]

Most patients improve after a kidney transplant. However, the hyperparathyroidism may persist in some patients. This is called tertiary hyperparathyroidism and may be irreversible and require a surgical intervention.

Generally, healthy women do not form cancellous bone on the endosseous cortical surface. This phenomenon also occurs in diabetes and subnormal parathyroid hormone secretion. This may be seen in ESRD patients with subnormal PTH levels, a condition known as adynamic bone disease. Low cancellous bone formation rate may not increase fracture risk since primary bone strength lies in the cortex. Low bone formation is associated with reduced skeletal buffering of calcium and increased soft tissue calcification. This is generally the final stage of skeletal adaptation to a surplus of calcium.[6] Parathyroid hormone can be monitored by an assay methodology test.

Immunoglobulin A (IgA) nephropathy is not well understood but there may be a genetic basis of the pathology.[5] IgA is deposited in the glomerulus of the kidney and causes dysfunction of the kidney.[14] A total of 25–50% of these patients progress to renal failure and end-stage renal disease.[11] There may be a genetic component to the cause of this disease.

Cigarette smoking is significantly associated with IgA nephropathy.[15] This patient smokes and declines to quit.

Cinacalcet (Sensipar) is an orally administered drug to treat secondary hyperparathyroidism in hemodialysis patients and parathyroid carcinoma.[16,17] Cinacalcet is not for treatment of primary hyperparathyroidism. It is a calcimimetic, it mimics serum calcium, normalizes it, and causes the parathyroid to reduce hormone secretion. It suppresses serum PTH levels and helps to manage serum calcium and phosphorus levels in

patients undergoing hemodialysis. The goal of therapy is to maintain a parathyroid serum level of 150–300 pico g/mL thus routine assessment of serum calcium, phosphorus, and PTH is important. Gastrointestinal discomfort is a significant side effect of cinacalcet that may require cessation of therapy. Other side effects include seizure, diarrhea, myalgia, and dizziness.

Chronic kidney disease can produce systemic chronic inflammation.[18] This is a factor in endothelial dysfunction, cardiovascular disease, hypertension, and malnutrition. Saliva is also involved. Salivary components can increase in concentration and flow rates decrease. Hyposalivation occurs frequently in hemodialysis patients and contributes to an increased caries rate. This patient had rampant caries. Cytokines occur in saliva and reflect the inflammatory status and general health of a patient. The cytokine profile in saliva is abnormal in periodontitis patients and smokers and is not well understood. Salivary cytokine flow is dramatically decreased in patients with chronic renal disease. The significance of this down regulation of inflammatory markers is not well understood.[18] It may be that these markers indicate risks for oral diseases.

These patients are administered heparin to prevent coagulation during dialysis. Heparin is a sulfated polysaccharide and accomplishes anticoagulation by inhibiting thrombin, prevents fibrin formation, activation of platelets, and activation of clotting factors V and VIII.[19] Local control was instituted with local anesthetic infiltration. To ensure bleeding control this patient was also administered a 6% tranexamic acid oral rinse. Tranexamic acid, an antifibrinolytic, acts by inhibiting plasmin and plasminogen and clotting factors V and VIII.[20] This may directly counteract heparin.

The estimated longevity of this patient may be 20–25 years, to the age of 55–60 years. However, future technology may prolong his life. Thus, this patient may enjoy many years of comfort and function with implant-supported complete dentures. His kidney transplant candidacy was delayed due to his severely carious teeth and associated chronic abscesses. Most of the teeth required extraction. Without appropriate treatment further deterioration and increased risk for acute infection would occur. Extraction and construction of removable prostheses subject the patient to osseous atrophy and inconvenience. Dental implant treatment offers the opportunity to preserve the osseous volume and freedom from caries.

Dental treatment

The patient discussed herein had rampant caries that rendered most of his teeth unrestorable.

This patient's main issue was dental caries and not periodontal bone loss. The most important issue related to implant treatment was the osseous support required for long-term success. Since new trabecular bone formation in this disease is not well calcified, there may be an expectation of a high rate of failure of osseointegration or failure under load. However, a rough implant surface may provide a substrate for osteoblasts, which may or may not encourage osteogenesis. An implant osseous mechanical failure may occur when there is a fracture of weak trabecular struts followed by a cascade of failure of the more substantial trabecular struts from overload. Treatment in experimental animals with parathyroid hormone, (iahPTH1-34), induced thicker trabecular struts and was able to prevent failure of subsequently placed implants.[21] However, this research was performed on rats and this outcome may not be extrapolated to human physiologic response. Additionally, primary stability of implants depends on cortical bone. Cancellous trabecular bone may not be as an important support mechanism for dental implants. Any patient may have osseous support that varies among an array of particular sites, which means that cortical and trabecular bone varies in thickness and mineralization and thus supportive qualities. Cortical thickness may be a more important parameter for dental implant support. However, if the cortical bone is losing thickness because of the disease and trabecular bone is formed preferentially but not being substantially mineralized then supportive implant osseointegration may be compromised. This may not be clinically evident until there is a late failure in the prosthetic phase. Using long and wide diameter implants distributes the per-square-mm force over a larger area of the weakened supporting bone and thus may reduce the stress on the bone resulting in attenuated strain making failure less likely.[22,23] The patient reported here is a young man and may live a substantial number of years. Extracting all of the unsalvageable teeth without implant placement would relegate him to a life of edentulism and removable prosthetics. Importantly however, cinacalcet therapy may eliminate or reduce the osseous consequences of the disease, allowing adequate and ongoing osseous implant support.

Another study in rats found that parathyroid hormone therapy was more effective for bone to implant regeneration in aged animals than in young animals.[24] The older animal had successful implant integration but under prosthetic loading the implant/s may potentially fail if mineralization is not substantial.

The patient's bone biopsy and radiographs appeared to have normal trabeculation and normal appearing radiodensity. The bone felt very dense and hard at the time of the initial extractions by the surgeon (DF). These parameters lend credence for adequate osseous implant support. Eventual kidney transplantation with a well-functioning graft usually reverses secondary hyperparathyroidism and may resolve the osseous dystrophy.

All the implants in this case presented were placed immediately into fresh extraction sockets without flaps being raised. Caneva and colleagues performed a comparison study of flapless versus flap closure of immediate implant placement in dogs.[25] They concluded that there was no prevention of bone resorption or alveolar dimensional changes. [25] However, in this study the flapless facial side gingiva was sectioned and the periosteal covering was lifted and disturbed. This probably compromised the osseous blood supply from the periosteum and may have induced resorption. Also, the flapless sites in the study were not covered with calcium sulfate or collagen thus exposing the osseous healing to oral conditions. In the study, the implants were placed at the level of the osseous crest and this may be inappropriate for bone healing. There may be 1–2 mm loss of bone height after an extraction. Additionally, the osseous healing may not be allowed to be covered by advancing epithelium forming a defect and thus there may be more bone resorption. Animal studies may not be extrapolated to human application due to the considerable

differences in the comparative physiological properties but these studies may give direction for human treatment.

Dietary magnesium deficiency may be implicated in late implant failure.[26] Animal studies show that dietary deficiencies may result in decreased trabecular bone, bone loss, and release of proinflammatory cytokines and alteration of parathyroid hormone.[26]

During the planning phase the mandibular anterior teeth were considered for nonextraction. They were extracted because of the high risk for caries in this patient and potential issues with the selected anterior guided occlusal scheme. According to experience of one of the authors (DF), a small number of natural teeth left in a multiple implant-supported treatment tend to develop overload issues and catastrophically fracture.

Anterior guidance was the selected occlusal scheme. During working excursion the natural mandibular cuspids would bear on the lingual aspect of the implant-supported maxillary cuspids the implant at site #12 was angled to the lingual to avoid an osseous dehiscence. An angled abutment was placed on this implant. Angled placed implants and angled abutments are appropriate to use in these situations. They fail at about the same rate as straight implants and abutments and the angulation microstrain on the bone may induce apposition.[13]

Full arch extraction and immediate implant placement has been previously reported.[27] Immediate loading in this case may have overloaded the provisional prosthesis and interrupted healing.

Polyvinyl siloxane impression material was used for its accuracy, nontoxicity to bone and very slight irritation to epithelium.[28]

The prostheses replaced first molar to first molar occlusion in both arches. Twelve prosthetic teeth in each arch is generally considered sufficient to maintain occlusal vertical dimension and appropriate masticatory function.[29]

Conclusions

After appropriate diagnosis, medical treatment and planning, implant treatment for patients with IgA nephropathy with secondary hyperparathyroidism and osteodystrophy may be successfully surgically and prosthetically treated with dental implants supporting fixed full-arch, fixed dental prostheses with an appropriate occlusal scheme. Appropriate calcium therapy should be instituted with timely serum assessments. It may be important to chemotherapeutically maintain the serum calcium to prevent inappropriate remodeling of the supporting bone in these patients.

Abbreviations

CKD: chronic kidney disease
ESRD: end stage renal disease
IgA: immunoglobulin A
iPTH: intact parathyroid hormone
PDL: periodontal ligament

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