

# Association between Dental Abscess and the Pathogenesis of Post-Streptococcal Glomerulonephritis: A Case Report

Safa Gadi<sup>1</sup>, Lojaen Sendy<sup>2</sup>, Faris Alotaibi<sup>3</sup>, Aboubakri Boucelham<sup>4</sup>

<sup>1</sup>BDS, SB-PD, <sup>2</sup>BDS, <sup>3</sup>BDS and <sup>4</sup>BDS, DESO, King Saud Medical City, Riyadh, Saudi Arabia

---

**Abstract:** Dental abscess is a frequently occurring infectious process known to the health practice. The reaction of the infection relay on the virulence of the bacteria, host resistance factors, and regional anatomy. Serious consequences arising from the spread of a Dental Abscess lead to significant morbidity and mortality. Acute dental abscess is Polymicrobial, comprising of strict anaerobes, such as anaerobic Cocci, Prevotella, Fusobacterium species, and Facultative anaerobes, such as viridans group Streptococci and the Streptococcus anginosus group. In the pathogenesis and evolution of Glomerular Nephropathies, localized infections play an important role. Attention is usually focused on upper respiratory tract infections (pharyngeal, tonsillar and sinus), less concern being given to dental foci. The incidence of Post Streptococcal Glomerulonephritis appears to be decreasing. Post Streptococcal Glomerulonephritis is caused by an infection with Streptococcus Bacteria. The infection causes blood vessels in the kidneys to develop inflammation; this block the renal organs ability to filter urine.

**Keywords:** Dental Abscess, Glumerulonephritis, Streptococcus and Kidney infection.

---

## I. INTRODUCTION

Dental abscess is a frequently occurring infectious process known to the health practice. The reaction of the infection relay on the virulence of the bacteria, host resistance factors, and regional anatomy. Serious consequences arising from the spread of a Dental Abscess lead to significant morbidity and mortality. Acute dental abscess is Polymicrobial, comprising of strict anaerobes, such as anaerobic Cocci, Prevotella, Fusobacterium species, and Facultative anaerobes, such as viridans group Streptococci and the Streptococcus anginosus group. <sup>[1]</sup> In the pathogenesis and evolution of Glomerular Nephropathies, localized infections play an important role. Attention is usually focused on upper respiratory tract infections (pharyngeal, tonsillar and sinus), less concern being given to dental foci. Several patients with IgA nephropathy have a chronic infection (e.g. dental abscess, chronic sinusitis or cryptic tonsillitis), approximately 40% of patients with IgA nephropathy experiencing recurrent macroscopic hematuria, which occurs within 48 hours of an infection. <sup>[2]</sup> The incidence of Post Streptococcal Glomerulonephritis appears to be decreasing. <sup>[3]</sup> Post Streptococcal Glomerulonephritis is caused by an infection with Streptococcus Bacteria. The infection causes blood vessels in the kidneys to develop inflammation; this block the renal organs ability to filter urine. <sup>[4]</sup> The typical presentation of Post Streptococcal Glomerulonephritis is a full-blown nephritic syndrome with oliguric acute renal failure. Most patients have milder disease, and subclinical cases are frequent. Patients with overt disease demonstrate gross hematuria characterized by red or smoky urine, headache, and generalized symptoms such as anorexia, nausea, vomiting, and malaise. <sup>[5]</sup> Swelling of the renal capsule can cause flank or back pain. Physical examination may report hypervolemia, edema, or hypertension. Coexisting rheumatic fever is highly unexpected. <sup>[6]</sup> Acute Post Streptococcal Glomerulonephritis usually is recognized on clinical and serologic grounds without the need for biopsy, specifically in children with a typical history. The comprehensive prognosis in classic Post Streptococcal Glomerulonephritis is excellent. Greater than 95% of patients retrieve immediately and recover to normal renal function within three to four weeks with no long-term squeals. <sup>[7]</sup>

Until now there has been inadequate data regarding the role of dental foci in the pathogenesis of Glomerular Nephropathies. These foci should be recognized and treated carefully under antibiotic protection, in a stable stage of the disease.

## II. CASE PRESENTATION

A 6 years old male was referred from Maternity and Children's Hospital – Buraidah, Qassim to King Saud Medical City - Riyadh with peri-orbital swellings, dark urine, vomiting and fever associated with history of Dental Abscess related to Maxillary Right Area and no recent history of upper respiratory tract infections. He was fully conscious with facial edema. Urine analysis showed RBC > 100/PHF, proteinuria, with granular casts. CBC showed WBC 11.590/ul, HgB: 10.8 g/dl, platelets: 374000/ul. U/E showed urea: 18.9 mmol/L (high), Creatinine: 261 umol/L (high). Abdominal ultrasound showed both kidneys enlarged in size with parenchymal bright echogenicity grade 1. Patient was diagnosed with Post Streptococcal Glomerulonephritis, he was admitted for 2 weeks and the treatment was started by Cefuroxime antibiotic, Hydralazine and Amlodipine as Anti-Hypertensive Medication. Then, Patient was referred to Pediatric Dental Clinic for Examination and Treatment. Upon oral examination: Poor Oral Hygiene, Multiple Carious Lesions and Deep Caries in Maxillary Right Second Primary Molar with Dental Abscess (Fig.1), radiographically: the Maxillary Right Second Primary Molar has Radiolucency in Interradicular area (Fig.2). Dental treatment was Extraction under Local Anesthesia of Maxillary Right Second Primary Molar (Fig.N). Post operative visit in Pediatric Hospital was after 1 month and the U/E showed urea: 4 mmol/L and Creatinine: 41 umol/L. and follow up in Pediatric Dental clinic showed Good Oral Hygiene and Good Healing of Extraction site.



Figure 1: Poor Oral Hygiene, Multiple Carious Lesions and Deep Caries in Maxillary Right Second Primary Molar with Dental Abscess.



Figure 2: The Maxillary Right Second Primary Molar has Radiolucency in Interradicular area.



Figure N: Extraction of Maxillary Right Second Primary Molar.

### III. DISCUSSION

The case presented with Dental Abscess associated with Post Streptococcal Glomerulonephritis. Despite the fact that pathogenesis of Glomerulonephritis remains unknown, various types of triggering factor have found to establish Glomerulonephritis. Endogenous mechanism, made up of autoimmunity and malignancies, and exogenous factors, for instance infectious organisms and drugs, have been accredited to the development of Glomerulonephritis. The pathway of Glomerulonephritis have been largely reviewed and analyzed by Chadban and Atkins<sup>[8]</sup> Cunard and Kelly<sup>[9]</sup> Naicker, antibody deposition within the kidney is well known to be the initial step of Glomerular inflammation in numerous forms of Glomerulonephritis, including Cryoglobulinemia and Goodpasture's disease. Dental Abscess pathogens are able to infiltrate the systemic circulation.<sup>[10]</sup> For example, viridians group Streptococci and the Streptococcus anginosus group has exhibit an invasive tendency toward endothelial cell cultures. In spite of, further studies on renal biopsy specimens to find specific Streptococcus bacteria or antigens seem to be conclusive.<sup>[11]</sup>

### IV. CONCLUSION

It is looks as that Dental Abscess may be an overlooked and treatable cause of Glomerulonephritis which call for more consideration. We suppose that the causative link between Dental Abscess disease and Glomerulonephritis are conceivable both invasions of the Glomeruli by Dental Abscess pathogens. Further investigations are advocated to be integrated in order to disclose the Dental Abscess pathogens and after Dental Abscess treatment in patients with unknown primary Glomerulonephritis.

### REFERENCES

- [1] Gluhovschi Gh, Trandafirescu V, Schiller A, Petrica L, Velciov S, Bozdog G, Bob F, Gluhovschi C. The significance of dental foci in glomerular nephropathies. *Medicine and Biology* Vol.10, No 2, 2003, pp. 57 – 61.
- [2] Lagrue G, Sadreux T, Laurent J, et al. Is there a treatment of mesangial IgA glomerulonephritis [letter]? *Clin Nephrol* 1980; 14: 161.
- [3] Nordstrand A, Norgren M, Holm SE. Pathogenic mechanism of acute post-streptococcal glomerulonephritis. *Scand J Infect Dis* 1999;31: 523-37.
- [4] Pan CG, Avner ED. Glomerulonephritis associated with infections. In: Kliegman RM, Stanton BF, St. Geme, JW III, Schor NF, eds. *Nelson Textbook of Pediatrics*. 20th ed. Philadelphia, PA: Elsevier; 2015:chap 511.
- [5] Brady HR, O'Meara YM, Brenner BM. Glomerular diseases. In: Kasper DL, et al., eds. *Harrison's Principles of internal medicine*. 16th ed. New York: McGraw-Hill, 2005:1674-94.
- [6] Sieck JO, Awad M, Saour J, Ali H, Qunibi W, Mercer E. Concurrent poststreptococcal carditis and glomerulonephritis: serial echocardiographic diagnosis and follow-up. *Eur Heart J* 1992; 13:1720-3.
- [7] Couser WG. Glomerulonephritis. *Lancet* 1999; 353:1509-15.
- [8] Chadban SJ, Atkins RC. Glomerulonephritis. *Lancet*. 2005; 365(9473): 1797–1806.
- [9] Cunard R, Kelly CJ. 18. Immune-mediated renal disease. *J Allergy Clin Immunol*. 2003;111(2 Suppl):S637–S644.
- [10] Tonetti MS, D'Aiuto F, Nibali L, et al. Treatment of periodontitis and endothelial function. *N Engl J Med*. 2007;356(9):911–920
- [11] Clynes R, Dumitru C, Ravetch JV. Uncoupling of immune complex formation and kidney damage in autoimmune glomerulonephritis. *Science*. 1998;279(5353):1052–1054