



The Lardaceous Slobber-Sialolipoma

Anubha Bajaj*

Department of Histopathology, Panjab University/A.B. Diagnostics, India

***Corresponding Author:** Anubha Bajaj, Department of Histopathology, Panjab University/A.B. Diagnostics, India.

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Lipoma emerges as a benign mesenchymal neoplasm configured of aggregates of mature adipose tissue. In contrast to lipomatosis, lipoma is a frequently discerned neoplasm. Generally, lipoma is associated with nonspecific clinical features. The essentially benign lipoma configured of accumulated mature adipose tissue is exceptionally encountered within salivary glands although parotid gland may be incriminated.

Initially scripted by Nagao in 2002, benign metamorphosis and impaction of salivary gland with mature adipose tissue engenders sialolipoma.

Sialolipoma represents as an uncommonly discerned variant of lipoma. Tumefaction is constituted of mature adipose tissue admixed with normal salivary gland elements as acinar cells, ductal epithelial cells, myoepithelial cells or basal cells. Sialolipoma may articulate intra-glandular or extra-glandular lesions.

Initially denominated by Yau in 1997, lipoadenoma represents as a gradually progressive neoplasm configured of glandular structures. Upon histological assessment, sertoliform morphology along with focal aggregates of mature adipose tissue may be delineated. Besides, foci of oncocytic metamorphosis and sebaceous differentiation may be encountered.

Lipoma configures as a commonly discerned, benign mesenchymal neoplasm incriminating major salivary glands. An estimated 3% of parotid tumours are exemplified by lipoma [1,2].

Tumefaction is incidentally discerned. Generally, individuals beyond >40 years are incriminated. Occasionally, paediatric subjects are implicated. A mild male predominance is encountered [1,2].

Sialolipoma exhibits chromosomal translocation t(12,14) or genomic rearrangements within HMGA gene [1,2].

Sialolipoma may represent a mean age of disease occurrence at 61 years although no age of disease emergence is exempt. Neoplasms confined to minor salivary glands demonstrate a female predilection.

Sialolipoma is commonly confined to parotid gland, submandibular gland, hard palate or soft palate [2,3].

Generally, neoplasm is engendered due to entrapment of salivary gland tissue within a lipoma configured of aggregates of mature adipose tissue. Clinical behaviour is benign. Tumefaction is devoid of lesion reoccurrence [2,3].

Cytological examination exhibits nonspecific features. Spindle cell lipoma or sialolipoma composed of spindle shaped cells demonstrates a bland cellular component wherein spindle shaped cells are interspersed within a myxoid background. Mature adipose tissue cells appear immune reactive to CD34+. Tumour cells are immune non reactive to S100 protein [2,3].

Upon gross examination, incriminated salivary gland delineates a well circumscribed tumefaction, reminiscent of lipoma confined to diverse soft tissue sites. Median tumour magnitude appears at 2 centimetres although neoplasm ranges from one centimetre to 4 centimetres in dimension [2,3].

Upon microscopy, sialolipoma is configured of a bland component of aggregates of mature adipose tissue commingled with normal salivary gland tissue, constituted of cellular component of acinar cells, ductal epithelial cells, basal cells or myoepithelial cells [3,4].

Sialolipoma is comprised of accumulated mature adipose tissue intermixed with normal salivary gland components as acinar cells, ductal cells, basal cells and myoepithelial cells [3,4].

Additionally, alterations such as duct ectasia with fibrosis, prominent lymphocytic infiltrate along with articulated nodular lymphoid aggregates confined to the stroma, oncocytic modifications or sebaceous differentiation may be delineated [3,4].

Sialolipoma may depict a distinctive vascular variant, designated as sialoangiolipoma, demonstrating commingled vascular articulations. Besides, variants such as osteolipoma may be encountered.

Lipoadenoma is preponderantly (>90%) constituted of mature adipose tissue cells intermingled with proliferating glandular articulations or configured, sharply defined duct - acinar units. Neoplastic cellular component appears reminiscent of sertoliform tubules. Additionally, foci of oncocytic metamorphosis, sebaceous differentiation or squamous metaplasia may be exemplified [3,4].

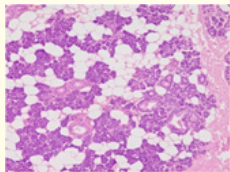


Figure 1: Sialolipoma delineating aggregates of mature adipose tissue cells intermingled with normal salivary gland component as acinar cells, basal cells, myoepithelial cells and ductal cells [6].

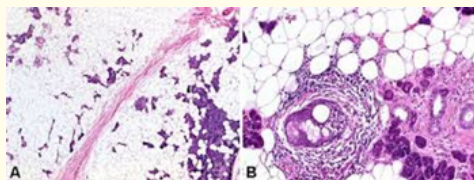


Figure 2: Sialolipoma demonstrating aggregates of mature adipose tissue cells commingled with normal salivary gland constituents as acinar cells, basal cells, myoepithelial cells and ductal cells [7].

Oncocytic lipoadenoma appears immune reactive to epithelial membrane antigen (EMA), cytokeratin, CK19, CK7, CK14, CK5/6, or alpha-1-antichymotrypsin [4,5].

Epithelial cell population confined to foci of oncocytic transformation demonstrates a dual cellular component. Ductal epithelial cells appear immune reactive to CK7 or CK19. Basal cells appear immune reactive to p63, CK14 or CK5/6 [4,5].

Tumour cells appear immune non reactive to calponin or actin.

Sialolipoma requires segregation from a pleomorphic adenoma as the neoplasm may exemplify extensive lipometaplasia or lipomatosis.

Upon T1 weighted and T2 weighted magnetic resonance imaging (MRI), lipoma manifests a signal intensity akin to circumscribing subcutaneous adipose tissue. Besides, fat suppression upon magnetic resonance imaging (MRI) can be beneficially adopted to assess lipomas of salivary gland.

Lipoma is traversed by fibrous tissue septa, especially lesions circumscribing vascular articulations [4,5].

Exceptional variants of lipoma depict a biphasic pattern wherein serous tissue appears diffusely disseminated amidst mature adipose tissue, thereby configuring a sialolipoma. Extraneous countenance of sialolipoma simulates normal parotid gland tissue. Sialolipoma is encapsulated and delineates a heterogeneous image due to soft salivary gland tissue commingled with mature adipose tissue [4,5].

Diffusion weighted magnetic resonance imaging (DWI) of lipoma exhibits specific range of mean apparent diffusion coefficient (ADC).

Sialolipoma can be appropriately subjected to simple surgical extermination of the neoplasm. Generally, tumour reoccurrence is absent [4,5].

Bibliography

1. Allen J, et al. "Multi-organ dysfunction in cerebral palsy". *Frontiers in Pediatrics* 9 (2021): 668544.
2. Koman LA, et al. "Cerebral palsy". *Lancet* 363 (2004): 1619-1631.
3. Colver AF and Sethumadhavan T. "The term diplegia should be abandoned". *Archives of Disease in Childhood* 88 (2003): 286-290.
4. Ehlert R, et al. "Cerebral palsy: Influence of TheraTogs on gait, posture, and functional performance". *Fisioter Movement* 30.2 (2017): 307-317.
5. El Shamy S, et al. "Efficacy of axial TheraTogs on gait pattern in children with dyskinetic cerebral palsy: a randomised controlled trial". *Bulletin of Faculty of Physical Therapy* 26 (2021): 12.

6. Stackhouse SK, et al. "Neuromuscular electrical stimulation versus volitional isometric strength training in children with spastic diplegic cerebral palsy: a preliminary study". *Neurorehabilitation and Neural Repair* 21 (2007): 475-485.
7. Van der Linden ML, et al. "Functional electrical stimulation to the dorsiflexors and quadriceps in children with cerebral palsy". *Pediatric Physical Therapy* 20 (2008): 23-29.
8. Durham S, et al. "Effect of functional electrical stimulation on asymmetries in gait of children with hemiplegic cerebral palsy". *Physiotherapy* 90 (2004): 82-90.
9. Dali C, et al. "Electrical threshold stimulation (TES) in ambulant children with CP: a randomised double-blind placebo-controlled clinical trial". *Developmental Medicine and Child Neurology* 44 (2002): 364-369.
10. Karabay İ, et al. "Short-term effects of neuromuscular electrical stimulation on muscle architecture of the tibialis anterior and gastrocnemius in children with cerebral palsy: preliminary results of a prospective controlled study". *American Journal of Physical Medicine and Rehabilitation* 94.9 (2015): 728-733.
11. Bohannon RW and Smith MB. "Inter-rater reliability of a modified Ashworth scale of muscle spasticity". *Physical Therapy* 67 (1987): 206-207.
12. Lundkvist Josenby A, et al. "Longitudinal construct validity of the GMFM-88 total score and goal total score and the GMFM-66 score in a 5-year follow-up study". *Physical Therapy* 89 (2009): 342-350.
13. Ko J and Kim M. "Reliability and responsiveness of the gross motor function measure-88 in children with cerebral palsy". *Physical Therapy* 93 (2021): 393-400.
14. Dawson N, et al. "Examining the reliability, correlation, and validity of commonly used assessment tools to measure balance". *Health Scientific Report* 1.12 (2018): 1-8.
15. El-Shamy SM. "Effects of antigravity treadmill training on gait, balance, and fall risk in children with diplegic cerebral palsy". *American Journal of Physical Medicine and Rehabilitation/Association of Academic Physiatrists* 96.11 (2017): 809-815.
16. Alabdulwahab SS. "Electrical stimulation improves gait in children with spastic diplegic cerebral palsy". *NeuroRehabilitation* 29 (2011): 37-43.
17. Flanagan A, et al. "Evaluation of short-term intensive orthotic garment use in children who have cerebral palsy". *Pediatric Physical Therapy* 21.2 (2019): 201-218.
18. Levitt S and Addison A. "Treatment of Cerebral Palsy and Motor Delay". 6th Edition. Hoboken, NJ: Wiley-Blackwell, (2019): 157-308.
19. Pool D, et al. "Effects of short-term daily community walk aide use on children with unilateral spastic cerebral palsy". *Pediatric Physical Therapy* 26.3 (2014): 308-317.
20. Bethoux F, et al. "Long-term follow-up to a randomized controlled trial comparing peroneal nerve functional electrical stimulation to an ankle foot orthosis for patients with chronic stroke". *Neurorehabilitation and Neural Repair* 29 (2015): 911-922.
21. Wright M and Wallman L. "Cerebral palsy". In: Campbell S, editor *Physical therapy for children*. Philadelphia: W.B Saunders (2012): 591-615.
22. Comerford MJ and Mottram SL. "Functional stability re-training: principles and strategies for managing mechanical dysfunction". *Manual Therapy* 6 (2001): 3-14.
23. Glaviano NR, et al. "Influence of patterned electrical neuromuscular stimulation on quadriceps activation in individuals with knee joint injury". *International Journal of Sports Physical Therapy* 9 (2014): 915-923.
24. Woollacott MH and Shumway-Cook A. "Postural dysfunction during standing and walking in children with cerebral palsy: what are the underlying problems and what new therapies might improve balance?" *Neural Plasticity* 12 (2005): 211-219; discussion 263-272.
25. Sterba J, et al. "Horseback riding in children with cerebral palsy: effect on gross motor function". *Developmental Medicine and Child Neurology* 44 (2000): 301-308.
26. Daichman J, et al. "The effects of a neuromuscular electrical stimulation home programme on impairments and functional skills of a child with spastic diplegic cerebral palsy: a case report". *Pediatric Physical Therapy* 15 (2003): 153-158.
27. Mittal R and Narkeesh A. "Review study on the effect of vestibular apparatus stimulation on postural muscle tone in cerebral palsy". *Journal of Exercise Science and Physiotherapy* 8 (2012): 11-19.