ORIGINAL RESEARCH PAPER

Pathology

CYTOHISTOPATHOLOGICAL CORRELATION OF GOUT IN A TERTIARY CARE CENTRE: A ONE-YEAR OBSERVATIONAL STUDY

KEY WORDS:

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INTRODUCTION

Gout is a crystal arthropathy caused by tissue deposition of needle-shaped, negatively birefringent MSU crystals in the setting of hyperuricemia. Polarised microscopy of synovial fluid is the reference standard; periarticular tophi are equally well sampled by FNAC or biopsy when fluid is unavailable. Indian comparative data on the two tissue methods in the same cohort are limited. This one-year tertiary-care study records cytohistopathological yield and its correlation with biochemical markers.

AIM AND OBJECTIVE-

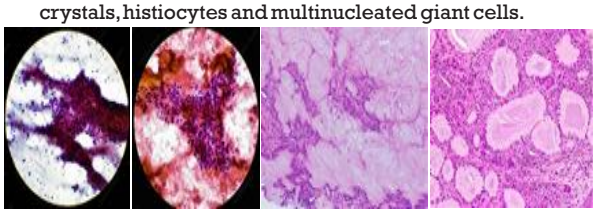
1. Document cytological and histological features of suspected gout.
2. Correlate morphology with serum urate and inflam-matory markers.
3. Compare diagnostic yield of FNAC versus histopathology.

MATERIALS AND METHODS

Observational study, Department of Pathology, Hi-Tech Medical College and Hospital, Bhubaneswar (2023–24). Twelve consecutive clinically suspected cases that had both FNAC and biopsy were included. FNAC (22–23 G) smears were stained with MGG and Pap and examined under compensated polarised light. Biopsies were processed routinely (H&E); unstained sections were polarised when crystals were suspected. Synovial fluid was analysed in cases with effusion. Serum uric acid, ESR, CRP and ASO titre were recorded in all. Demonstration of needle-shaped negatively birefringent crystals, with or without a giant-cell reaction, was diagnostic. Scanty FNAC was coded as inadequate, not negative.

RESULTS

- Twelve patients; mean age 54 years (range 42–66); 10 males, 2 females. Serum uric acid was raised in every case.
- FNAC demonstrated MSU crystals in 10/12 (83.3%). Two aspirates were inadequate (scanty material). Adequate smears showed chalky granular debris, needle-shaped crystals, histiocytes and multinucleated giant cells.



- Histopathology confirmed crystal deposits in 11/12 (91.7 %), typically as pale amphophilic material rimmed by palisaded histiocytes and foreign-body giant cells, with variable chronic inflammation.

Table 1. DiagnosticYield

Modality	n / 12	%
FNAC — MSU crystals	10	83.3
Histopathology — crystal deposits	11	91.7
Synovial fluid analysis	9	75.0
Raised serum uric acid	12	100

- Synovial fluid agreed with histology in 9/12 (75%). ESR and CRP were raised in most patients. ASO titre exceeded 166 Todd units in three cases (6, 7, 12) and was treated as incidental; morphology remained that of gout.

Table 2. Inflammatory Markers

Case	ESR (mm/h)	CRP (mg/L)	ASO (Todd U)
1	45	12.4	125
2	50	18.2	145
3	38	9.8	123
4	60	22.5	137
5	55	15.3	132
6	42	7.9	168*
7	68	25.1	170*
8	47	10.2	129
9	53	14.6	139
10	35	8.5	142
11	62	27.8	133
12	40	6.7	167*

**ASO >166 Todd units (conventional upper limit). These three values did not change the morphological diagnosis.*

DISCUSSION

MSU crystal demonstration remains the only definite diagnostic criterion. In this series histopathology (11/12) outperformed FNAC (10/12); both FNAC misses were technical (scanty aspirate), a recognised limitation of fibrotic tophi. Adequate FNAC smears recapitulate the same crystal and giant-cell mor-phology and remain an appropriate first-line test for accessible swellings. Raised ESR/CRP reflect inflammatory load and Isolated ASO elevation in three patients is non-specific and should not divert the diagnosis when crystals are documented.

LIMITATIONS:

small single-centre sample (n = 12); synovial fluid not obtained in every case; formal sensitivity/specificity agai-nst an independent gold standard was not calculated. Aqueous processing can dissolve urate; parallel smear examination and polarised microscopy were used to reduce this artefact.

CONCLUSION

Histopathology had a higher diagnostic yield than FNAC. FNAC is useful when cellular; an inadequate aspirate warrants

biopsy. Combined cytohistopathology and biochemistry is the practical diagnostic algorithm in tertiary-care.

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