**Background:** Thrombotic microangiopathies (TMA) are rare group of heterogenous disorders that ultimately result in endothelial and vasculature damage. The pathophysiology behind TMA is diverse: some patients have inherited complement disorders, for others, infection, pregnancy and drugs can precipitate its onset [1]. Despite growing interest in the molecular basis underpinning TMA and potential therapeutic targets, there are currently no published studies examining the frequency or characteristics of thrombotic microangiopathy in an unselected population.

 **Method:** Retrospective case-note review. All biopsies collected at our centre from 25/4/97 to 10/10/16, with acute or chronic features of TMA, as described by the KDIGO consensus [2] were included.

**Results:** 227/6033 native renal biopsies had features of TMA. 212 biopsies were available for analysis: 6 were excluded as they were referred slides, 8 were from patients who had more than one biopsy and we were unable to locate 1 biopsy.

The median age at diagnosis was 51 years (IQR 38-63) and 136/212 patients had an identifiable contributing cause (secondary TMAs, see table to right).

66/76 (88%) of patients with idiopathic TMA had histological features of active TMA (as defined by current KDIGO criteria [2]).

The median serum creatinine at the time of biopsy for this cohort was 260umol/L (IQR 160-500).

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| --- | --- | --- | --- | --- |
|  | **Total** | **Secondary TMA** | **Idiopathic TMA** | **P value** |
| Hypertension specified on biopsy request | 66/206 | 14/130 (6 unknown) | 52/76 | <0.0001 |
| Male sex | 122/212 | 71/136 | 51/76 | 0.0426 |

**Conclusion:** We have identified all biopsies in an unselected population with features of TMA and found the general incidence to be 3.76%. The majority of patients (64%) have a known associated secondary cause. However in approximately 1/3 of patients, no secondary cause is identifiable. These are predominantly younger male patients and over 2/3 of these patients were hypertensive at the time of biopsy. In these cases, TMA is not a benign disease. Initial review would suggest it is associated with progressive renal dysfunction and a high incidence of end stage renal disease.

**References:**

1. Barbour T, Johnson S, Cohney S et al (2012). Thrombotic microangiopathy and associated renal disorders. Nephrol Dial Transplant. 27(7):2673-85.

2. Goodship T, Cook H, Fakhouri F et al (2017). Atypical haemolytic uraemic syndrome and C3 glomerulopathy: conclusions from a “Kidney Disease: Improving Global Outcomes” (KDIGO) Controversies Conference. Kidney Int. 91(3):539-551