had endoscopy within 24 hours. The median time-to-endoscopy for the sub group of COVID positive patients (n=6) was 17.5 hours (IQR16). A chi-squared test was used to compare the likelihood of those with COVID undergoing endoscopy <24 hours, compared to those who were COVID negative/unknown, and there was no significant difference (1.080 p=0.299).

38 re-bleeds occurred, and 37 deaths. COVID19 positivity was associated with a significantly increased risk of death when reviewed with a Chi squared test (4.46 p=0.035). However, COVID19 positivity was not associated with any increased risk of re-bleeding (0.009 p=0.922).

The number of cases of AGIB, and monthly median time to endoscopy, varied considerably but there was a strong correlation between the two over the 14 month study period, see figure 1. The month of April 2020 had the lowest number of cases at only 6, a figure that is an outlier at >1.5 standard deviations from the mean for the study period. Reductions in the incidence of AGIB seemed to coincide with national lockdowns.

Conclusion Patients with COVID19 and AGIB had a high risk of death, even though the data would suggest this death was not from re-bleeding. It is more likely that AGIB is a sign of physiological strain from COVID 19 infection.

We showed that AGIB cases reduced markedly at the first lockdown, which suggests that patients were not willing (or perhaps able) to present to secondary care services. This pattern was replicated during lockdowns 2 and 3. However, further review over a broader time period would be required, to further explore this observed phenomenon.

If we are using the 'time-to-endoscopy' as a surrogate marker of standards of endoscopy services, our trust appears to have offered its best AGIB service during this first lockdown. This may represent reduced strain on the service. We were able to offer a comparable time-to-endoscopy to those patients who were COVID positive. The overall median time to endoscopy was comparable to the figure of 21.2 hours, found by *Siau et al.* (*Siau et al.*, 2019) in the multi-site audit of 2017.

Abstract PTH-89 Figure 1

Learning from how our services have performed in this pandemic will help us prepare ourselves in case of future similar challenges.

REFERENCE

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Pancreas and neuroendocrine

PTU-67 DIFFERENTIAL EXPRESSION OF THE POTASSIUM OF VOLTAGE-GATED CHANNEL SUBFAMILY MEMBERS, KCNH

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Introduction Human pancreatic cancer is an intractable malignancy and is the seventh leading cause of cancer deaths worldwide and the fifth leading cause in the UK. Pancreatic cancer has ranked the 11th most common cancer in the world and the UK. The incidence and mortality of pancreatic cancer are linked with aging and is more favorable in men than in women. The dominant risk factors of pancreatic cancer remain vague and the 5-year survival rate remains poor (~9%) despite clinical advances. Voltage-gated potassium (Kv) channels are known to modulate varied functions of epithelial cells in pancreas and its ion transportation, a function partly regulated by calmodulin related proteins. The dysregulation or alterexpression of potassium channels in pancreatic cancer has yet been fully characterised. In this study, we evaluated the transcript expression levels of KCNH1, KCNH2 and KCNH3 in pancreatic cancer and their clinical and prognostic significance.

Methods Pancreatic tissues including tumour tissue sections and their adjacent normal tissue sections from a cohort of patients in our lab (n=223). The gene transcripts of the three KCNH members were quantified in the tissues and analyses were carried out against the clinical, pathological and outcome factors of the patients.

Results We find that there is a significant difference in the expression of KCNH1 and KCNH2, but not KCNH3 in pancreatic tumour tissues compared with normal tissues p=0.011 for KCNH1 and p=0.015 for KCNH2). Overall, adenocarcinomas exhibited higher levels of all three KCNH than ductal carcinomas of the pancreas. KCNH members do not appear to have a clearly link to the staging of pancreatic cancer and only showed a weaker link to the survival of the patients. In our cohort database, we have identified that All three KCNH were found to have significantly correlated with a tumour repressor molecule, MTSS1/MIM (Metastasis Suppressor-1/Missing In Metastasis) (r=0.602, 0.469 and 0.508 for KCNH1, KCNH2 and KCNH3, respectively, p<0.0001 by Spearman test). It was further demonstrated that KCNH1 and MTSS1/MIM collectively is a risk factor for the death of the patients (HR 1.607, p=0.017) in predicting pancreatic cancer related survival (Mean survival 15.8±2.6 months versus 28.1±3.8 months for patients with high and low aberration of KCNH1 and MTSS1, p=0.008).

Conclusion KCNH family is aberrantly expressed in clinical pancreatic cancer and together with MTSS1/MIM offer a prognostic factor for the survival of the patients.

PTU-68 THE EXPRESSION AND CLINICAL SIGNIFICANCE OF MLN64 IN HUMAN PANCREATIC CANCER

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Due to submitting loading errors, the fully list of authors and the institutions of this abstract were incomplete. The following are the correct information for the authors and institutions:

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