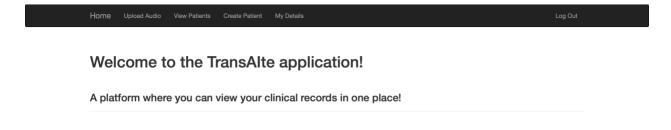
# User Manual of the Transl'AI'te Web Application (Doctor)

### Case 1. View Patients' Consultation Records



# 2. Click **View Patients** on the top bar.



#### 3. You can View all Patients' Records now!



#### 4. Click the View Records button to View specific Patients' Record!

Viewing Siddharth Chaudhary's Past Clinical Documents

| name | appointment date | clinical specialty | download transcribed audio | download summarised report |
| example.txt | 2022-03-01 | Neurology | Download | Download |

Home Upload Audio View Patients Create Patient My Details

#### Consultation Report

First Name: Siddharth
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Date Of Birth: 2001-12-21
Email: sidd640@icloud.com

#### **Transcribed Consulation:**

Therapeutic inhibition of tumour angiogenesis and multiple signalling pathways associated with tumour development (e.g., pathways controlled by vascular endothelial growth factor receptors (VEGFRs) and platelet-derived growth factor receptors (PDGFRs)) results in clinically meaningful antitumor activity, as demonstrated across multiple tumour types, including colon cancer, pancreatic carcinoma, renal cell carcinoma (RCC), breast cancer (BC) and non-small-cell lung cancer (NSCLC) (HurwitzNone, 2005;SandlerNone, 2006;ManegoldNone, 2008;EscudierNone, 2009). When combined with chemotherapy, antiangiogenic therapies may provide effective treatment of historically treatment-resistant solid tumours (HurwitzNone, 2005;SandlerNone, 2006;MillerNone, 2007;ManegoldNone, 2008). Thus, treatment that specifically interrupts tumour vasculature through inhibition of various important receptor tyrosine kinase (RTK) signalling pathways, combined with a chemotherapy, may be of interest. Sunitinib malate, an oral multitargeted inhibitor of VEGFRs, PDGFRs, stem cell factor receptor (KIT), and other RTKs (AbramsNone, 2003;MendelNone, 2003;O'FarrellNone, 2003;Ac) specifically interrupts tumour development (ERC), in a provention of various insulations and tumour, and progressive, well-differentiated pancreatic neuroendocrine tumours (DemetriNone, 2006;MotzerNone, 2007;KulkeNone, 2008;RaymondNone, 2011;SUTENT (sunitinib malate) prescribing information (2012)). In phase I and It trials, sunitinib has also shown antitumor activity in patients with other advanced solid tumours, including BC, NSCLC, neuroendocrine tumour, sarcoma, thyroid cancer and melanoma (RosenNone, 2003;FaivreNone, 2006;BursteinNone, 2008;BocinskiNone, 2008). Gemcitabine is a nucleoside analogue that primarily targets cells undergoing DNA synthesis (S-phase) and also blocks progression of cells through the G1/S-phase boundary. Gemcitabine is used (alone or in combination with other chemotherapies, such as cisplatin or carboplatin) across a broad spectrum of solid tumou

#### **Summarized Consultation Report:**

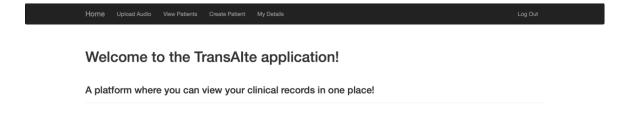
Extensive preclinical evidence suggests additive and/or synergistic effects in solid tumour models when a variety of chemotherapies, including gemcitabine, are combined with targeted agents, including sunitinib (YeeNone, 2004;CarterNone, 2007;ChristensenNone, 2008), as demonstrated in a recently reported phase I trial of sunitinib on a continuous daily dosing schedule plus gemcitabine in patients with advanced solid tumours (BrellNone, 2012). The phase I dose-finding study reported here was also conducted to investigate the safety, pharmacokinetics (PK) and antitumor activity of sunitinib (on an intermittent dosing schedule) in combination with gemcitabine in patients with advanced solid tumours for whom curative therapy was not available. Schedule 4/2 was not evaluated past the initial combination dose level (sunitinib 37.5 mg and gemcitabine 750 mg m-2) as it proved to be an awkward scheduling regimen in practice because of missed or delayed doses of gemcitabine (see Determination of MTD). Two of elevant patients (18%) on Schedule 2/1, pre-amendment, had DLTs at the dose level of sunitinib 37.5 mg-gemcitabine 750 mg m-2(appendicitis/abscess and QTc prolongation). Schedule 4/2 was not pursued beyond the initial dose level of sunitinib 37.5 mg plus gemcitabine 750 mg m-2for practical reasons (i.e., because of missed or delayed doses of gemcitabine because of slow recovery from neutropenia). In the earlier phase I trial with gemcitabine, growth factor support was excluded and the recommended phase II dose was gemcitabine 675 mg m-2on days 1 and 8 and sunitinib 25 mg on continuous daily dosing (BrellNone, 2012). Our results also suggest that sunitinib combined with gemcitabine may be a more active regimen than gemcitabine-based chemotherapy, which has also shown limited activity in sarcomatoid RCC (StadlerNone, 2003;NanusNone, 2004).



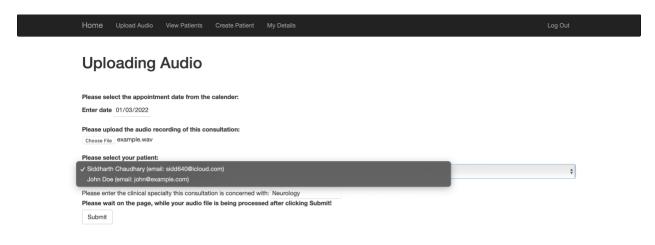
## Case 2. Upload Consultation Audio File



# 2. Click Upload Audio on the top bar.

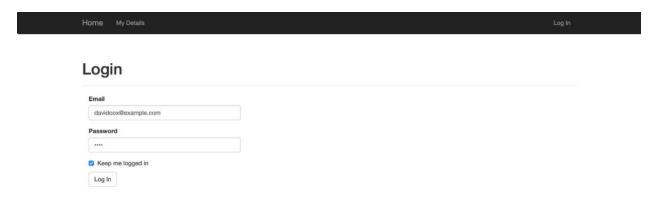


### 3. Upload the audio for **One Specific Patient!**

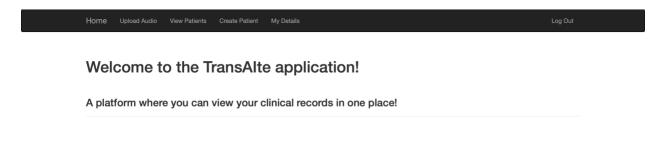


4. Check all the details and Click **Submit.** File will be uploaded and records will be generated. You can now view patients' records.

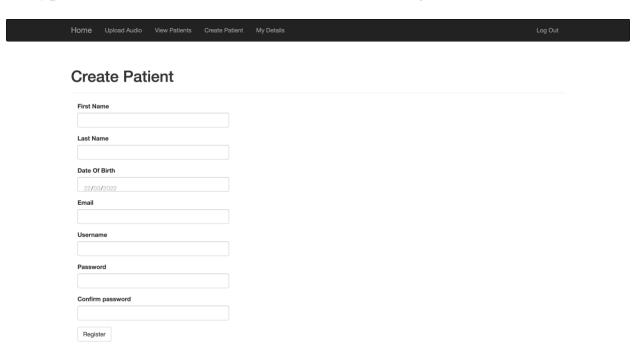
#### **Case 3. Create a Patient Account**



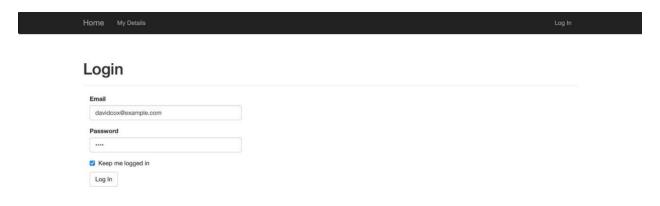
# 2. Click **Create Patient** on the top bar.



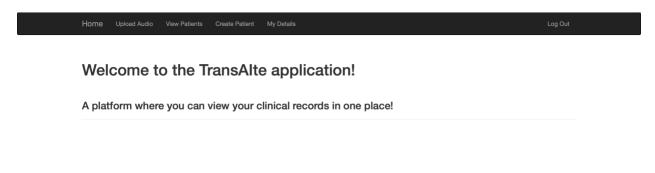
# 3. Type in Information of the Patient and Click Register



# Case 4. View My Detail



# 2. Click My Details on the top bar.



# 3. You can View your Details now!

